Impact of Nuclear Weapons

ARMY FIELD MANUAL 8-9,
NAVY MEDICAL PUBLICATION 5059,
AIR FORCE JOINT MANUAL 44-151

CHAPTER 1

GENERAL INFORMATION

101. Purpose of AMedP-6(B), Part I.

The purpose of this publication is to provide medical personnel of the NATO Armed Forces with information on the biomedical effects of nuclear weapons and the impact of the use of nuclear weapons on the different aspects of medical field operations.

102. General Impact of Nuclear Weapons.

a. Total nuclear war with utilization of all available nuclear weapons could result in complete devastation of the involved nations' military combat and logistic systems as well as their supporting civilian social structures and economies. (See paragraphs 625 and 631.) However, situations short of total nuclear war are possible in which nuclear weapons could be employed in limited numbers or for a limited time, along with conventional weapons. Under such circumstances, effective military operations could continue and would require the continuing support of an effective medical service.

b. It is essential that medical personnel at all levels be prepared for the problems associated with limited nuclear warfare. This handbook has been prepared to provide those responsible for medical support planning, training, and field operations with specific information critical to the understanding and solution of these special problems. The subjects covered in this publication are many and varied, reflecting the complexities involved in the nuclear sciences and the varied needs of the people for whom this book was prepared. Accordingly, the subject matter presented includes discussions of atomic structure and radioactivity, characteristics of nuclear detonations, descriptions of the factors related to the diagnosis, treatment and prognosis of nuclear warfare casualties, and guidance applicable to the organization and operation of medical units on a nuclear battlefield. In addition, information has been included on the special problems associated with nuclear accidents in peacetime.

CHAPTER 2

CONVENTIONAL AND NUCLEAR WEAPONS - ENERGY PRODUCTION AND ATOMIC PHYSICS

SECTION I - GENERAL

201. Introduction.

As a first step in developing an understanding of the medical implications of nuclear warfare, it is essential to understand how a nuclear weapon differs from a conventional high explosive weapon. Accordingly, a comparison will be made in this chapter between the mechanisms of energy production in conventional and nuclear detonations. In addition, certain principles of atomic structure and physics are presented to aid in the understanding of these differences.

SECTION II - MECHANISMS OF ENERGY PRODUCTION

202. Definition of Explosion.

An explosion can be described as the sudden release of large amounts of energy within a limited space as the system involved is converted to a more stable one. The basic laws of thermodynamics pertaining to the conservation of energy require that energy must be released when a system is converted to another of greater stability, i.e., one containing less energy.

203. Conventional Chemical Explosion.

a. The molecules of conventional chemical explosives are considered to be in a high-energy or unstable state. When such a system is made to react, products of greater stability are formed and energy is released. With a conventional explosive, such as trinitrotuluene (TNT), the energy is derived from a sudden, violent chemical reaction, altering various bonds between the molecules of the explosive's chemical compounds, i.e.,

2 Molecules TNT + Heat = Reaction Products + Energy

31.3×10^{-19} joules (7.5×10^{-19} Cal (net)).

The amount of energy released in such a reaction is directly proportional to the difference between the total binding energy contained within the initial, unstable

system and that contained within the final, more stable system. This net energy release is called the heat of explosion.

b. As in all chemical reactions, mass and energy are conserved separately; i.e., by the best methods of measurement available, the total mass and the total energy, including the heat of explosion, are found to be exactly the same, respectively, before and after the explosion.

204. Nuclear Detonations.

Energy released in a nuclear explosion is not produced by chemical reactions. Rather, it results from so-called nuclear reaction, fission and fusion, in which fundamental changes occur in the composition of the nuclei of the reacting material rather than in the electron shells as is the case in chemical reactions. In these nuclear reactions mass is actually converted to energy, and the amount of energy produced is many orders of magnitude greater than that available from chemical reactions. To fully appreciate the nature of these reactions, certain basic concepts related to atomic structure and nuclear reactions must first be understood.

205. Elements and Atomic Structure.

a. *Elements*. All substances are composed of one or more of over 100 different kinds of basic materials known as elements. There are 92 naturally occurring and at least 11 artificially produced elements, ranging from the simplest and lightest naturally occurring element hydrogen to the heaviest artificial element lawrencium.

b. *Atomic Structure*. The simplest structural unit of any element that can exist, while still retaining the chemical and physical characteristics of the element, is called an atom. An atom is composed of a central nucleus containing most of its mass and electrons orbiting in shells around the nucleus (Figure 2-I). The nucleus consists of a number of fundamental particles, the most important of which are the protons and neutrons.

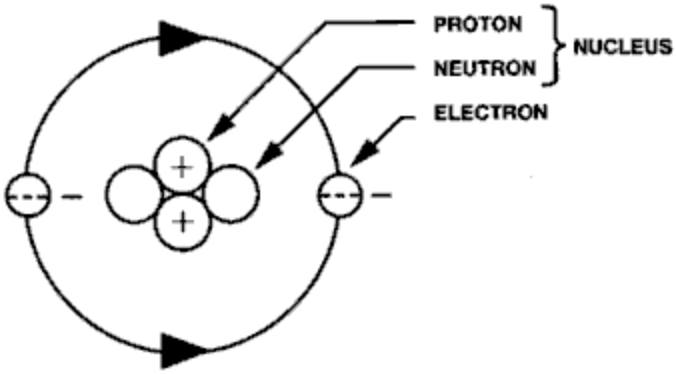


Figure 2-I. A Typical Atom

(1) The proton is a particle having a positive charge, equal in magnitude and opposite in sign to that of the electron. The proton's mass is approximately 1845 times greater than that of the electron.

(2) The neutron is an uncharged particle having a mass slightly greater than that of the proton, approximately equal to the sum of the masses of a proton and an electron.

(3) Electrons are negatively charged particles. They orbit the nucleus at discrete energy levels referred to as electron shells.

c. *Electrical Charge*. Atoms are electrically neutral when the number of negatively charged electrons orbiting the nucleus equals the number of positively charged protons within the nucleus. When the number of electrons is greater than or less than the number of protons in the nucleus, atoms are not electrically neutral and carry a net negative or positive charge. They are then termed ions and are chemically reactive, tending to combine with other ions of opposite net charge. When atoms are combined in molecules, they may share electrons to achieve stability of electron shell structure (Figure 2-II).

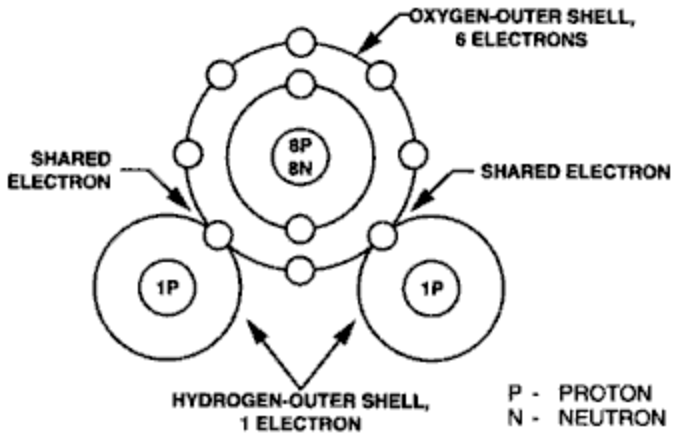


Figure 2-II. Illustration of a Molecule (H_20)

206. Isotopes.

a. Atoms of different elements have different numbers of protons in their nuclei. The term atomic number describes the number of protons in a nucleus. Although all the nuclei of a given element will have the same atomic number, they may have different atomic masses because they may contain different numbers of neutrons. Generally, this does not affect the chemical properties of the different atoms since the numbers of protons are not changed but does have profound effects upon nuclear stability of the different atoms. The total number of protons and neutrons in an atomic nucleus is referred to as the atomic mass number. Atomic species which have identical atomic numbers but different atomic mass numbers are called isotopes (Figure 2-III).

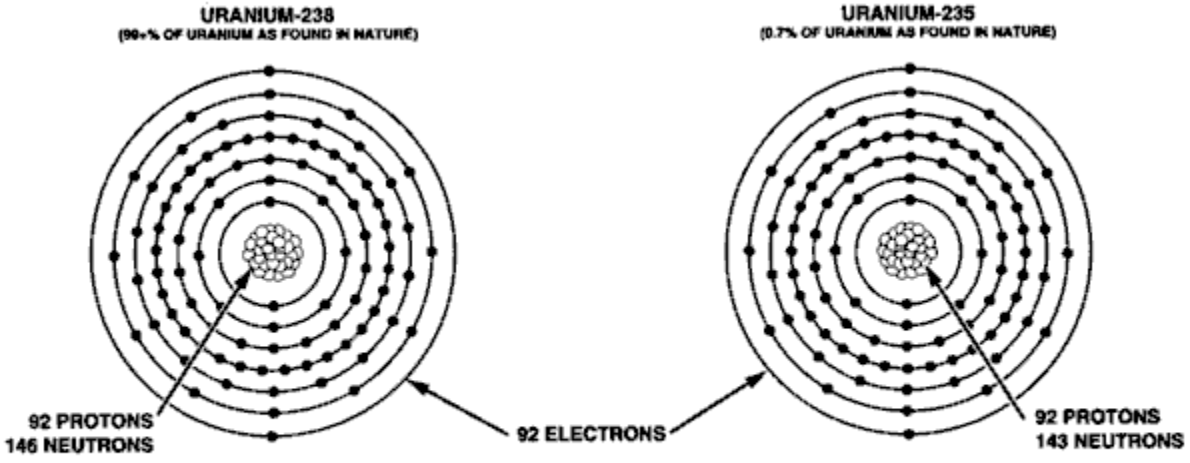


Figure 2-III. Isotopes of Uranium

b. The stable isotopes of elements have very definite ratios of neutrons to protons in their nuclei. As atomic mass numbers increase, the ratio of neutrons to protons increases according to a definite pattern (Figure 2-IV). If isotopes vary from this pattern, they are relatively unstable.

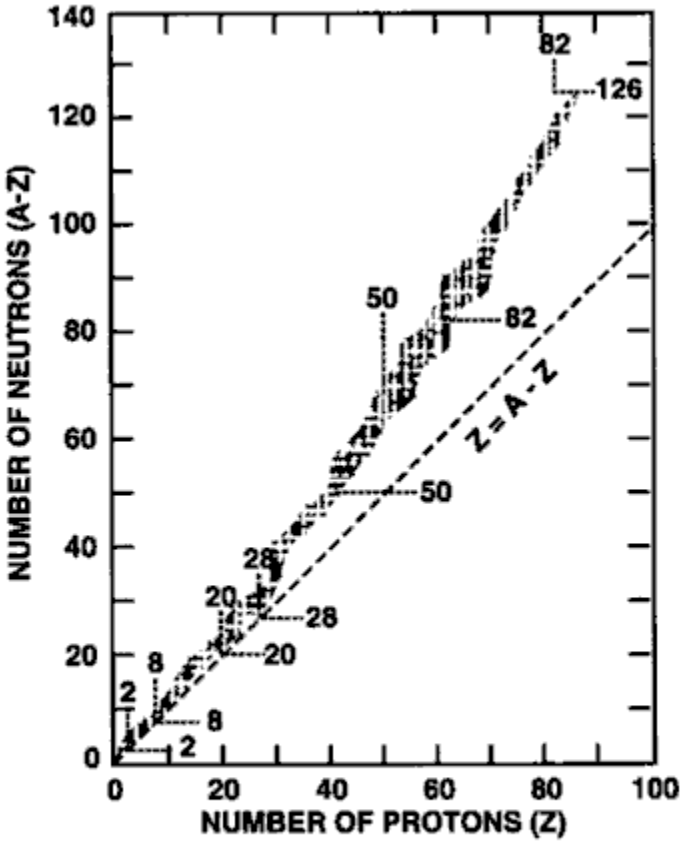


Figure 2-IV. Neutron to Proton Ratios

207. Atomic Mass Unit.

a. Common units of mass, such as grams, are much too large to conveniently describe the mass of an atomic nucleus or any of its constituent parts. To solve this problem a new unit was defined: the atomic mass unit (amu). The atomic mass unit is a relative unit defined arbitrarily by assigning a mass of 12 amu to the neutral atom carbon-12, the common isotope of carbon. One atomic mass unit equals 1.66×10^{-24} grams. Employing this value, the masses of the fundamental particles of an atom have been determined to be:

(1) Proton mass: 1.00727 amu.

(2) Neutron mass: 1.00867 amu.

(3) Electron mass: 0.00055 amu.

b. Logically, it should be possible, knowing the number of particles comprising a particular atom, to calculate the mass of that atom. However, experiments have shown that the total mass of an atom is less than the sum of the masses of the atom's electrons, protons, and neutrons. For example, the measured mass of the isotope fluorine-19 atom is 18.99840 amu, while the sum of the masses calculated for the individual particles of that atom is 19.15708 amu. The difference of 0.15868 amu between the measured and calculated mass of the fluorine-19 atom is defined as the mass defect (Figure 2-V).

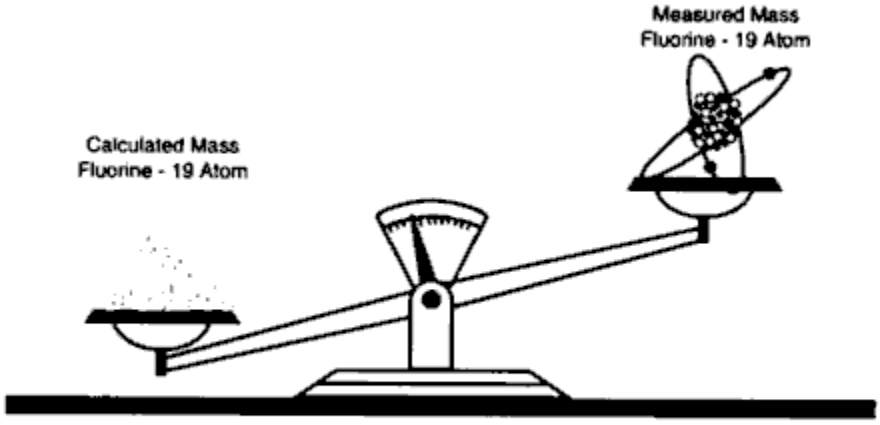


Figure 2-V. Illustration of a Mass Defect

c. Careful experimentation and study have shown that while the mass defect is real, the law of conservation of mass has not been violated. When basic particles combine to form an atom, a certain amount of mass is lost through conversion into energy in accordance with Einstein's equation $E = mc^2$, where **E** is the energy, **m** is the mass, and **c** is the velocity of light in a vacuum. The converted energy is considered to be binding energy, i.e., energy necessary to hold the nucleus together.

208. Symbols and Notation.

a. A standard notational form is used to identify the individual isotopes of a given element. The standard notation takes the following form:

A

$$^{A}_{Z}X$$

where X = chemical symbol of the element, Z = atomic number, and A = atomic mass number.

b. An example of the standard notation would be:

$$^{235}_{92}U$$

c. Reference to a chart of the nuclides would reveal that the element with an atomic number of 92 is uranium, the chemical symbol for which is U. The atomic mass number ^{235} identifies a uranium isotope having 92 protons and 143 neutrons (235 - 92 = 143) in its nucleus. Thus the isotope identified by the example notation is the naturally occurring, readily fissionable isotope of uranium used in nuclear weapons. The atomic number is frequently left off, and such an isotope may then be represented only by its mass number and chemical symbol, i.e., ^{235}U.

209. Fission.

a. Fission is a nuclear process in which a heavier unstable nucleus divides or splits into two or more lighter nuclei, with the release of substantial amounts of energy. The materials used to produce nuclear explosions by fission are those isotopes of uranium or plutonium which undergo fission most readily. These are ^{235}U and ^{239}Pu. When as illustrated in Figure 2-VI, a free neutron of the proper energy is captured by the nucleus of a fissionable atom, the resulting unstable nucleus will "split" producing two or more fission products (atoms of different elements formed from the protons, neutrons, and electrons originally comprising the nucleus before its fission), two or three free neutrons and a tremendous amount of energy.

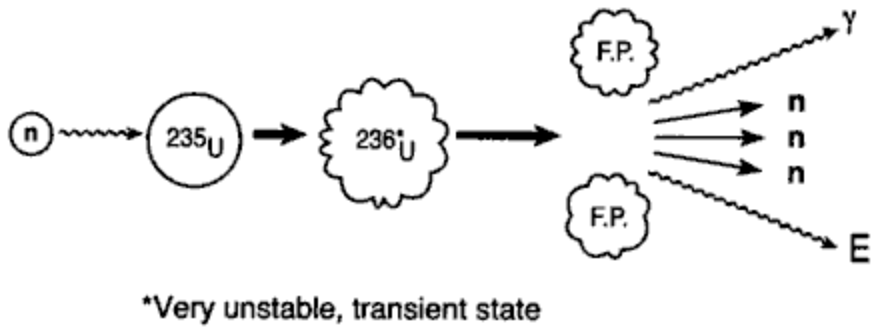


Figure 2-VI. Fission Process

b. In terms of continued energy production, the most significant point about the fission process is the emission of free neutrons, which can in turn produce other fission events, which in turn produce still another generation of free neutrons. Each generation of fission produced neutrons can produce a large number of fissions; and so, within a few generations, the total number of fissions produced can be tremendous.

c. While in principle a single neutron could initiate a chain reaction of nuclear fissions which could ultimately result in the splitting of each fissionable atom in a given mass, not all of the neutrons produce more fissions. Some of the neutrons may escape from the fissionable mass. Others may be removed by nonfission reactions. To initiate a chain reaction, sustain that reaction for a period sufficiently long to permit a buildup of explosive energy, and confine the released energy for as long as possible to maximize the weapon's explosive effect requires that a variety of special conditions be met.

210. Critical Mass.

The first prerequisite to be met in producing a fission-type nuclear explosion is that there must be enough material present and in the right configuration so that successive generations of neutrons can cause equal or increased numbers of fissions. The amount capable of sustaining a continuous or chain reaction is termed a critical mass.

a. Although fission events release more than 2 million times more energy per event than do chemical reactions, there still must be a tremendous number of fissions to result in the release of a significant amount of energy. To meet this requirement, a mass of fissionable material having specific characteristics must be assembled. Depending on size, and other factors to be discussed, a given mass of fissionable material may support one of three types of chain reactions:

(1) *Subcritical Chain Reaction*. A reaction in which the number of neutrons decreases in succeeding generations, thus not continuing.

(2) *Critical Chain Reaction*. A reaction in which the number of neutrons remains constant in succeeding generations.

(3) *Supercritical Chain Reaction*. A reaction in which the number of neutrons increases in succeeding generations.

b. To produce a nuclear explosion, a weapon must contain an amount of uranium or plutonium that exceeds the mass necessary to support a critical chain reaction, i.e., a supercritical mass of fissionable material is required. Several methods can be used to make a mass of fissionable material supercritical.

(1) The active material can be purified to eliminate unwanted chemical impurities that might otherwise absorb neutrons.

(2) Fissionable material can be enriched, i.e., the amount of ^{235}U as compared to ^{238}U can be increased.

(3) The material can be machined into the most efficient shape. A spherical shape can be employed to provide the greatest volume with the least surface area, thereby reducing the probability of neutron loss.

(4) Moderators can be used to slow down fission neutrons, increasing the probability of their producing fissions.

(5) Finally, neutrons that have escaped the active material can be reflected back by using suitable materials as reflectors. Reflectors, used as tampers, can also physically delay the expansion of the exploding material allowing more fission to occur thereby resulting in an increase in explosive energy.

c. Because of the stray neutrons produced in the environment by spontaneous fission, those present in the atmosphere from cosmic ray interactions as well as others generated in various ways, a critical or supercritical mass would be likely to melt or possibly explode. It is necessary, therefore, that, before detonation, a nuclear weapon contain no piece of fissionable material as large as a critical mass. At the time of the detonation, some method must be employed to make the mass supercritical by changing its configuration. Two general methods have been developed for quickly converting a subcritical mass into a supercritical one.

(1) In the first, two pieces of fissionable material, each less than a critical mass, are brought together very rapidly to forma single supercritical one. This gun-type assembly may be achieved in a tubular device in which a high explosive is used to blow one subcritical piece of fissionable material from one end of the tube into another subcritical piece held at the opposite end of the tube (Figure 2-VII).

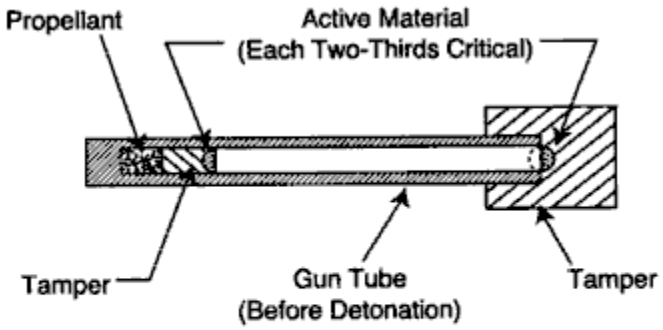


Figure 2-VII. Gun Assembly Principle

(2) In the second or implosion-type assembly method (see Figure 2-VIII), a subcritical mass of ^{235}U or ^{239}PU is compressed to produce a mass capable of supporting a supercritical chain reaction. This compression is achieved by the detonation of specially designed high explosives surrounding a subcritical sphere of fissionable material. When the high explosive is detonated, an inwardly directed implosion wave is produced. This wave compresses the sphere of fissionable material. The decrease in surface to volume ratio of this compressed mass plus its increased density is then such as to make the mass supercritical. An enhanced radiation (ER) weapon, by special design techniques, has an output in which neutrons and x-rays are made to constitute a substantial portion of the total energy released. For example, a standard fission weapon's total energy output would be partitioned as follows: 50% as blast; 35% as thermal energy; and 15% as nuclear radiation. An ER weapon's total energy would be partitioned as follows: 30% as blast; 20% as thermal; and 50% as nuclear radiation. Thus, a 3-kiloton ER weapon will produce the nuclear radiation of a 10-kiloton fission weapon and the blast and thermal radiation of a 1-kiloton fission device (Figure 2-IX). However, the energy distribution percentages of nuclear weapons are a function of yield.

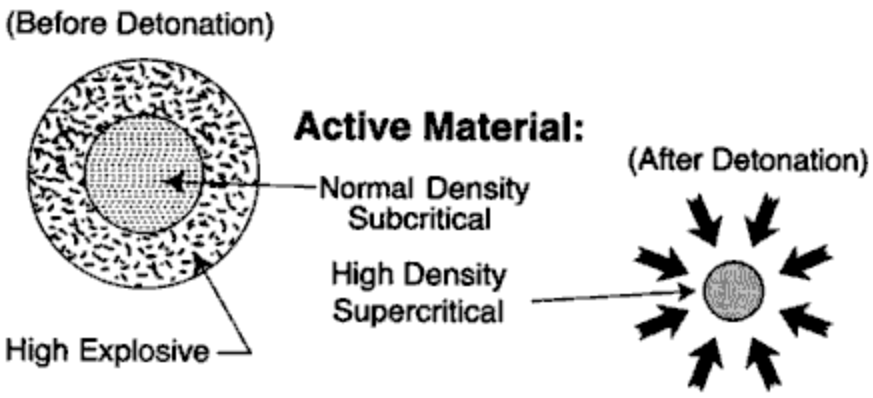


Figure 2-VIII. Implosion Assembly Principle

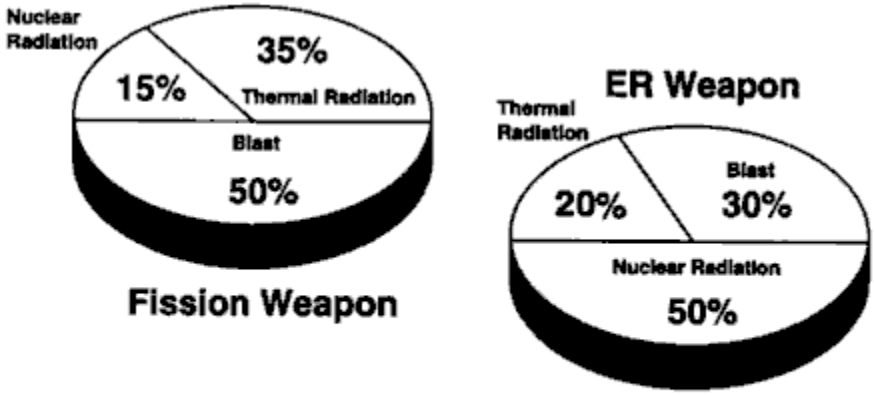


Figure 2-IX. Weapon Energy Distribution

211. Fusion.

In general, fusion may be regarded as the opposite of fission. It is the combining of two light nuclei to form a heavier nucleus. For the fusion process to take place, two nuclei must be forced together by enough energy so that the strong, attractive, short-range, nuclear forces overcome the electrostatic forces of repulsion. The two conditions necessary for the fusion of appreciable numbers of nuclei are high temperatures to accelerate the nuclei and high pressure density to increase the probability of interaction. The only practical way to obtain the temperatures and pressures required is by means of a fission explosion. Consequently, weapons with fusion components must contain a basic fission component. The energy released in the explosion of a fission-fusion weapon originates in approximately equal amounts from the fission and fusion processes.

SECTION III - RADIOACTIVITY AND NUCLEAR RADIATION

212. General.

Paragraph 208c. described the isotope ^{235}U as being "... the naturally occurring, readily fissionable isotope of uranium..." An expanded, but more complete, description would also have identified the isotope ^{235}U as being radioactive. Similarly, in a fission reaction most, if not all, of the fission products produced are radioactive.

213. Radioactivity.

The nuclei of certain naturally occurring isotopes, and of others produced artificially, contain excess energy, i.e., they are unstable. To attain stability, nuclei with excess energy emit that energy in the form of nuclear, ionizing radiation and, in that process, frequently change into different elements. (See paragraph 215e.) (Ionizing radiation is defined as radiation capable of removing an electron from a target atom or molecule, forming an ion pair.) Isotopes, the nuclei of which emit ionizing radiations to achieve stability, are termed radioactive. Radioactive isotopes are referred to as radioisotopes or radionuclides.

a. *Radioactive Decay*. The process wherein radionuclides emit ionizing radiation is also termed radioactive decay. Each radioisotope has its own characteristic decay scheme. A decay scheme identifies the type or types ionizing radiation emitted; the range of energies of the radiation emitted; and the decaying radioisotope's half-life.

b. *Half-Life*. Half-life is defined as the time required for half of the atoms of a given sample of radioisotope to decay. Half-life values range from fractions of a millionth of a second to billions of years. Theoretically, no matter how many half-lives have passed, some small number of nuclei would remain. However, since any given sample of radioactive material contains a finite number of atoms, it is possible for all of the atoms eventually to decay.

c. *Data Plotting*. Radioactive decay may be plotted in a linear form as shown in Figure 2-X or in a semilogarithmic form as in Figure 2-XI. The latter has the advantage of being a straight lineplot. The straight line form is used extensively in radiation physics, particularly when dealing with isotopes with short half-lives, since it allows direct determination by simple inspection of the activity at any given time with a precision adequate for most purposes.

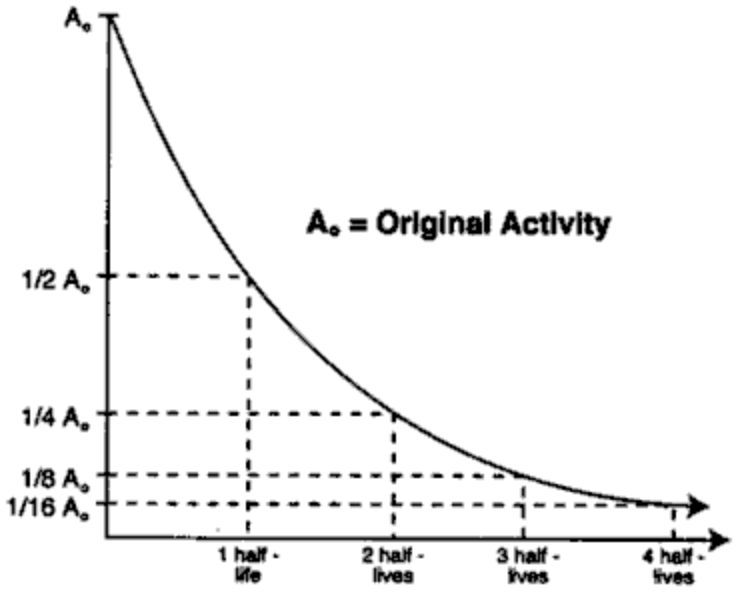


Figure 2-X. Radioactive Decay Plotted in Linear Form

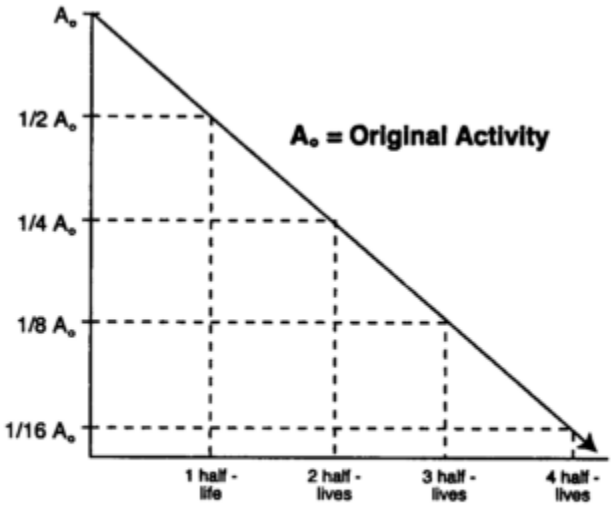


Figure 2-XI. Radioactive Decay Plotted in Semilogarithmic Form

214. Measurement of Radioactivity.

a. The international system of units is based on the meter, kilogram, and the second as units of length, mass, and time, and is known as Systems International (SI). The amount of radioactivity in a given sample of radioisotope is expressed by the new Systems International (SI) unit of the Becquerel (Bq). The old unit was the Curie (Ci).

One Becquerel of a radioisotope is the exact quantity that produces one disintegration per second. The Curie is 3.7×10^{10}Bq disintegrations per second. Thus 1 Bq = 2.7×10^{-11}Ci and 1 Ci = 3.7×10^{10}Bq. As the Becquerel is inconveniently small for many uses as was the Curie inconveniently large, prefixes such as micro (μ) (10^{-6}), milli (m) (10^{-3}), kilo (k) (10^{3}), mega (M) (10^{6}), and giga (G) (10^{9}) are routinely used. Following nuclear detonations, the amounts of radioactive material produced are very large and the terms pets-becquerel (PBq) (10^{15}Bq) and exabecquerel (EBq) (10^{18}Bq) may be used. The term megacurie (MCi) (10^{6}Ci) used to be used.

b. The amount of radioactive material available at any time can be calculated by using a specific mathematical formula:

$$A_t = A_0 e^{(-\lambda t)}$$

from which the following can be derived

$$A_t = A_0 e^{\left(\frac{-0.693t}{T_{1/2}}\right)}$$

since

$$\lambda = \frac{-0.693t}{T_{1/2}}$$

c. The terms in these formulae are as follows:

(1) A_t = activity remaining after a time interval, t.

(2) A_o = activity of sample at some original time.

(3) e = base of natural logarithms (2.718...).

(4) λ = decay constant of the particular isotope, derived from the half-life.

(5) t = elapsed time.

(6) $T_{1/2}$ = half-life of the particular isotope.

d. This formula can be used to calculate the activity (A) of an isotope after a specific time interval (t) if the half-life ($T_{1/2}$) and the original activity (A_o) are known.

(1) Example: If 3.7×10^{10}Bq (= 1.0 Ci) of ^{60}Co (cobalt) is the original amount of radioactive material at time t_o, what will be the activity of the ^{60}Co remaining 1 month later?

A1 month = activity remaining after 1 month (t)

A_0= 3.7 x = 10 ^{10}Bq (original activity)

$T_{1/2}$ = 5.27 years (half-life of ^{60}Co is 5.27 years)

t = 1 month (time elapsed since the original time).

(2) Substituting in the formula gives the following:

$$A_{1\ month} = 3.7 \times 10^{10} Bq\ e^{\left(\frac{0.693t}{5.27yr} \times 1\ month\right)}$$

(3) All values have to be converted to the same time units, in this case, years. Therefore:

$$A_{.0883yr} = 3.7 \times 10^{10} Bq\ e^{\left(\frac{0.693t}{5.27yr} \times .0833yr\right)}$$

$$= 3.7 \times 10^{10} Bq\ e^{\left(\frac{0.0577}{5.27yr}\right)}$$

$$= 3.7 \times 10^{10} Bq\ e^{(-0.0109)}$$

$$= 3.7 \times 10^{10} Bq\ e\ 0.99$$

(4) In other words, the activity of ^{60}Co after 1 month is 0.99 of its original activity, a reduction of only 1%. This could not be determined with precision from a graphic plot of activity versus time.

215. Nuclear Radiation.

Radioisotopes of heavy elements such as radium or uranium characteristically decay by emission if ionizing radiation in the form of alpha particles. Some heavy elements also decay by spontaneous fission which results in neutron releases. For the lighter elements, emission of beta particles is common. In addition, emissions of gamma or x-ray photons almost invariably accompany both alpha and beta particle radiation. This is important since gamma or x radiation constitutes the principal casualty producing form of ionizing electromagnetic radiation associated with nuclear explosions. X-ray and gamma photons are essentially identical, differing only in their points of origin. Gamma photons originate in the nuclei of decaying atoms while x- rays originate in

the electron shells surrounding nuclei. Refer to paragraphs 503-506 for detail of penetration capabilities of the types of radiation.

a. Even though they possess no net electrical charge, gamma and x-ray photons interact with atoms to produce ionization. Gamma photons have discrete energies over a very wide range, but are considerably less ionizing than alpha or beta particles but much more penetrating.

b. An alpha particle is a helium nucleus consisting of two protons and two neutrons all strongly bound together by nuclear forces. Alpha particles have a mass about 7000 times that of electrons and are ejected from the nuclei of radioactive atoms with one, or at the most several, characteristic and discrete energies. Although highly ionizing, alpha particles are only slightly penetrating.

c. Beta particle decay involves the conversion of a neutron into a proton and electron within the nucleus. While the proton is retained in the nucleus, the beta particle (electron) is ejected with a velocity dependent upon its kinetic energy. Opposed to alpha particles, beta particles show a continuous energy spectrum. Because of its smaller mass and relatively higher emission energies, a beta particle is less ionizing than an alpha particle but more penetrating.

d. In a fission process, neutrons are also released and consequently, make up a significant portion of the total radiation output.

e. From the discussion in paragraphs 215b and 215c, it can be seen that, depending upon the type of particulate radiation emitted in decay, decaying nuclei can, in addition to changing their energy states, be transformed into new elements. Examples of the transformation resulting from alpha and beta particle decay are shown in Table 2-I.

Table 2-1. Radioactive Decay

Isotope	Half-Life	Radiations Emitted	Decay Product	Half-Life
a. Fissionable Material				
Uranium-235	7.1×10^8 yr	α, γ	Thorium-231*	25.2 hr
Uranium-238	4.5×10^9 yr	α, γ	Thorium-234*	24 days
Plutonium-239	2.4×10^4 yr	α, γ	Uranium-235*	7.1×10^8 yr
b. Fission Products				
Lanthanum-140	40 hr	β^-, γ	Cerium-140	Stable
Iodine-131	8 days	β^-, γ	Xenon-131m	11.9 days
Strontium-90	28.9 yr	β^-	Yttrium-90	64 hr
Cesium-137	30.0 yr	β^-, γ	Barium-137m	2.5 min
c. Other Radioisotopes				
Radon-222	3.8 days	α, γ	Plononium-218*	3 min
Potassium-40	1.3×10^9 yr	β^-, β^+, γ	Cesium-40	Stable
			or Argon-40	Stable
Sodium-24	15 hr	β^-, γ	Magnesium-24	Stable
Hydrogen-3 (Tritium)	12.3 yr	β^-	Helium-3	Stable

* Includes other daughter radionuclides.

216. Interaction With Matter.

a. Ionizing radiation interacts with matter in one of two ways. It is either scattered or absorbed. Both result in deposition of energy in the target system. The mechanisms of absorption are of particular interest because:

(1) Absorption in body tissue may result in physiological injury.

(2) Absorption is a phenomenon upon which the detection of ionizing radiation is based.

(3) The degree of absorption or type of interaction is a primary factor in determining shielding requirements.

b. Transfer of energy from an incident photon or particle to the atoms of an absorbing target material may occur by several mechanisms.

(1) *Excitation*. This process involves the addition of energy to an atomic or molecular system, thereby transferring it from its ground or stable state to an excited or unstable state. Depending upon the type of interaction, either the atomic nucleus or one of its orbital electrons may absorb the excitation energy.

(a) Electron excitation occurs when relatively small amounts of energy are transferred. Here the electrons may only be moved to a higher energy level in the atom (Figure 2-XII).

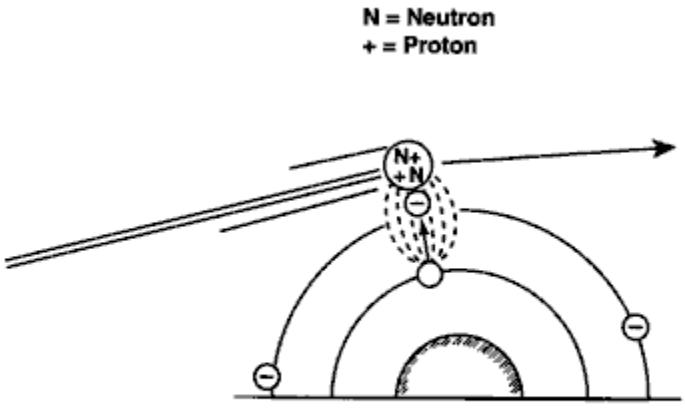


Figure 2-XII. Excitation of an Electron

(b) An excited electron will not retain its energy but will tend to return to its original energy level either by emitting the excess energy in the form of a photon of electromagnetic radiation (x-ray) or by transferring its energy to the electrons of other atoms or molecules.

(2) *Ionization.* As indicated previously, ionization is any process which results in the removal of an electron (negative charge) from an atom or molecule thereby leaving the atom or molecule with a net positive charge. Ionization occurs if alpha or beta particles, or gamma photons transfer sufficient energy to dislodge one of the electrons from the outer orbital shells of the target atom. Each ionization event produces an ion pair consisting of a free electron and the positively charged remainder of the atom (Figure 2-XIII).

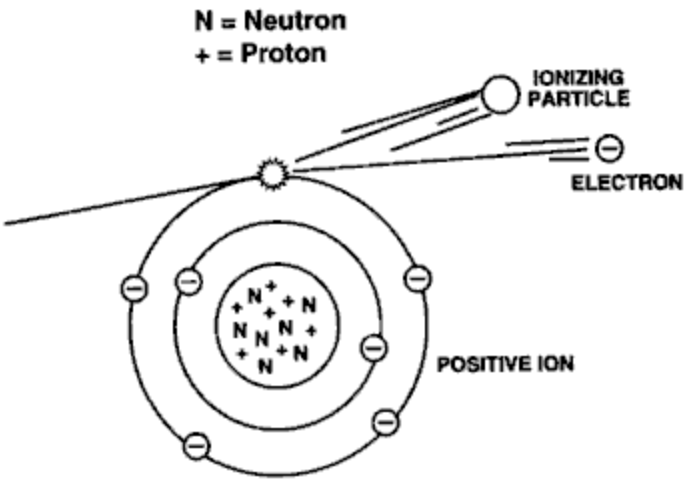


Figure 2-XIII. Electron Removal by Ionization

217. Gamma Interaction.

In terms of ionization, gamma radiation interacts with matter via three main processes: photoelectric effect, Compton scattering, and pair production.

a. *Photoelectric Effect*. This describes the case in which a gamma photon interacts with and transfers all of its energy to an orbiting electron, ejecting that electron from the atom (Figure 2-XIV). The kinetic energy of the resulting photoelectron is equal to the energy of the incident gamma photon minus the binding energy of the electron. The photoelectric effect is thought to be the dominant energy transfer mechanism for x-ray and gamma ray photons with energies below 50 keV (thousand electron volts), but it is much less important at higher energies.

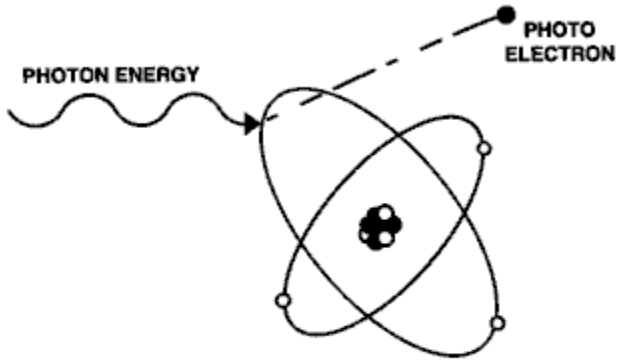


Figure 2-XIV. Gamma Interaction by Photoelectric Effect

b. *Compton Scattering.* This is an interaction in which an incident gamma photon loses enough energy to an orbital electron to cause its ejection, with the remainder of the original photon's energy being emitted as a new, lower energy gamma photon with an emission direction different from that of the incident gamma photon (Figure 2-XV). The probability of Compton scatter decreases with increasing photon energy. Compton scattering is thought to be the principal absorption mechanism for gamma rays in the intermediate energy range 100 keV to 10 MeV (million electron volts), an energy spectrum which includes most gamma radiation present in a nuclear explosion. Compton scattering is relatively independent of the atomic number of the absorbing material.

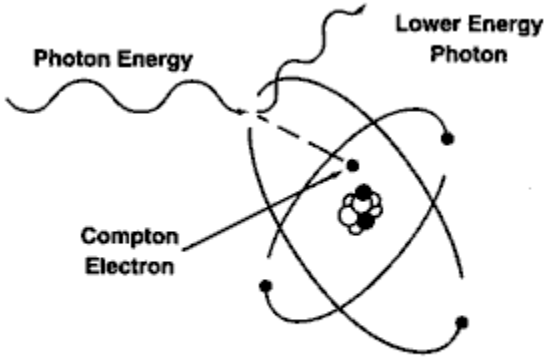


Figure 2-XV. Gamma Interaction by Compton Scattering

c. *Pair Production.* By interaction in the vicinity of the coulomb force of the nucleus, the energy of the incident photon is spontaneously converted into the mass of an electron-positron pair. A positron is a positively charged electron. Energy in excess of the equivalent rest mass of the two particles (1.02 MeV) appears as the kinetic energy of the pair and the recoil nucleus. The electron of the pair, frequently referred to as the secondary electron, is densely ionizing. The positron has a very short lifetime. It combines with 10-8 seconds with a free electron. The entire mass of these two particles is then converted to two gamma photons of 0.51 MeV energy each (Figure 2-XVI).

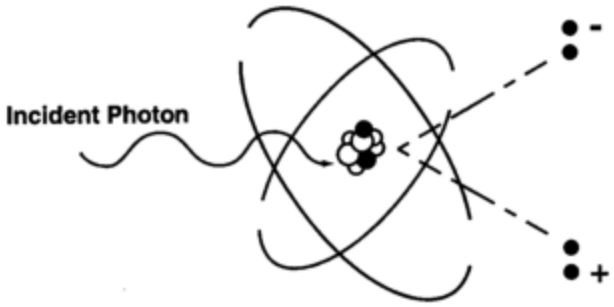


Figure 2-XVI. Gamma Interaction by Pair Production

218. Interaction of Charged Particles.

a. Due to the high probability of interaction between an alpha particle and orbital electrons in an absorbing medium, a large number of ion pairs are formed per unit path length. Since a finite fraction of the total kinetic energy of an alpha particle is absorbed with the formation of each ion pair, the alpha particle will lose its energy over a relatively short distance. For these reasons, the range of alpha particles is much less than the range of beta particles or gamma photons.

b. Beta particles and orbital electrons have negative charges, resulting in electrostatic repulsion when in the vicinity of one another. But, a beta particle has a charge opposite to that of the atomic nucleus, resulting in electrostatic attraction. Normally a beta particle loses its energy in a large number of ionization and excitation events in a manner analogous to the alpha particle. However, the range of the beta particle is considerably greater than that of an alpha particle. The beta particle travels longer distances between interactions and follows a "drunken man's path" through matter.

219. Specific Ionization.

a. The penetrating ability of radiation depends on the rate at which the radiation deposits energy along its path. The term specific ionization, which is defined as the average number of ion pairs generated per unit length of path, is used to describe the ionizing capability of ionizing radiations.

b. Generally speaking, the ion density along the path of a low-energy particle is greater than that along the path of a high-energy particle of the same mass and charge. This is because the low-energy particle is moving slower and has more time to interact. Its total pathway is shorter, however, and the total number of interactions may well be less. Likewise, the ion density towards the end of the path of a particle is greater than at the beginning, because its velocity is less and the probability of interaction is increased accordingly. Alpha particles are capable of producing the

highest specific ionization followed in order by beta particles and the secondary electrons produced by gamma-photon interactions (Table 2-II).

Table 2-II. Specific Ionization of Radiation

Radiation	Range in air	Speeds	Specific ionization
Alpha	5 - 7 cm	3,200 - 32,000 km/sec	20,000 - 50,000 ion pairs/cm
Beta	200 - 800 cm	25 - 99% speed of light	50 - 500 ion pairs/cm
Gamma	Use of half-thickness	Speed of light 300,000 km/sec	5 - 8 ion pairs/cm

c. The more common basis for comparing the various types of radiations is known as Linear Energy Transfer (LET), and represents the average energy released (or lost) per unit track length in ionization and excitation interactions. LET is usually expressed in units of KeV (thousands of electron volts) per micron of path length. To a considerable extent, the Relative Biological Effectiveness (RBE) of various radiations depends on the rate of energy loss (LET) along the paths of the individual ionizing particles or photons. Radiations with low LET such as x- or gamma rays produce diffuse ionizations throughout the medium. In contrast, the LET associated with neutrons or alpha particles is so high that the passage of a single track will, in all probability, put enough ionizations into a traversed cell to produce death.

220. Stopping Power.

The maximum ranges of ionizing radiation in matter depend not only on the characteristics of the specific particles or photons but also on the stopping power of the absorbing material. The stopping power of a material is a function of electron density or the number of electrons per unit volume of the substance and represents the total energy lost in collision and radiative interaction. The electron density increases as the density electrons per gram (10^{23}). Materials differ in their stopping power on the basis of the ratio of their atomic number to their atomic mass (Z/A) times the density of the material. The range of a charged particle in an absorbing material is inversely proportional to this ratio. For example, if the range of a given energy beta particle is 1 cm in water, its range in air would be much greater (about 10 m), and in iron much smaller (about 1 mm).

221. Half-Value Layer.

a. The concept of stopping power is not generally used in connection with material interactions of either gamma photons or uncharged neutrons. Since high-energy radiation is in general more penetrating than low-energy radiation, the specification of half-value layer (HVL) is often a convenient method of characterizing the penetrating quality of an energy spectrum. Half-value layer is defined as that absorber thickness which reduces a given radiation intensity to one-half of the incident value (Figure 2-XVII). Refer to Table 7-II for half-value layer thickness of common materials.

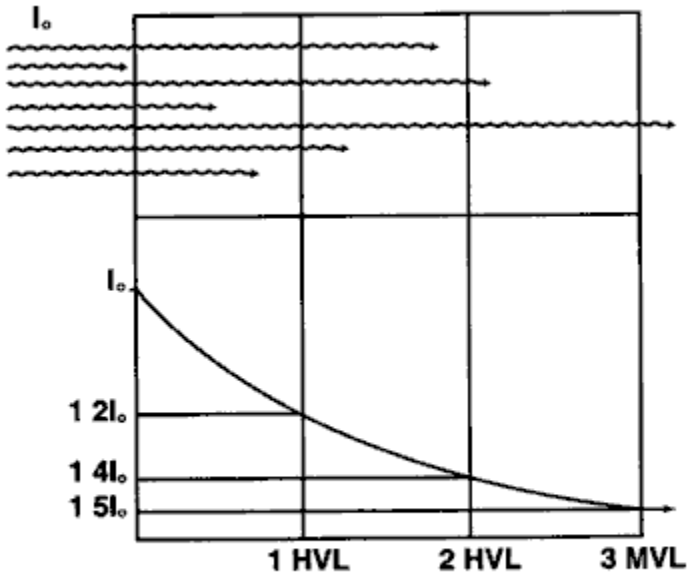


Figure 2-XVII. Attenuation of Gamma Radiation

b. The relatively high penetrating power of x and gamma radiation compared with charged particles is related to the fact that the absorption interactions are fairly rare occurrences. The attenuation of gamma radiation as it passes through matter can expressed by the following formula:

$$I = I_0 e^{(\mu_0 d)}$$

where:

(1) I = gamma radiation intensity after passing through a target material thickness d (see below).

(2) I_0 = intensity of the incident gamma radiation at the surface of the target.

(3) μ_0 = material-dependent attenuation coefficient (cm^{-1}).

(4) d = thickness of the target material (cm).

222. Neutron Interaction.

Although most ionizing radiation injuries associated with nuclear warfare will be attributable to gamma radiation previously described, a sufficient number of high-energy fission neutrons escape from the detonation to represent a significant hazard at considerable ranges.

a. The neutron is a particle and thus is fundamentally different from electromagnetic radiation. It also differs from other particulate radiations (alpha and beta) in that neutrons do not carry any electrical charge. As a result, neutrons do not interact with the orbital electrons of atoms. Instead, they interact directly with the nuclei of atoms, particularly those having low atomic mass numbers.

b. Depending on their point of origin, neutrons may have energies ranging from a fraction of an electron volt (Ev) for so-called thermal neutrons to several megaelectron volts (MeV) for fast (fission) neutrons, to fusion neutrons which have energies of up to 14 MeV (e.g., deuterium-tritium reaction). Most neutrons produced in a nuclear fission detonation will have energies less than 1 MeV. A small fraction will have energies above 3 MeV. In enhanced radiation weapons, there will be a preponderance of 14 MeV neutrons.

c. Neutrons transfer their energy to target atoms and molecules by elastic and inelastic collisions with nuclei.

223. Elastic Collisions.

In elastic collisions, part of the neutron's energy is transferred to the target atom in a manner analogous to a purely mechanical collision process (Figure 2-XVIII).

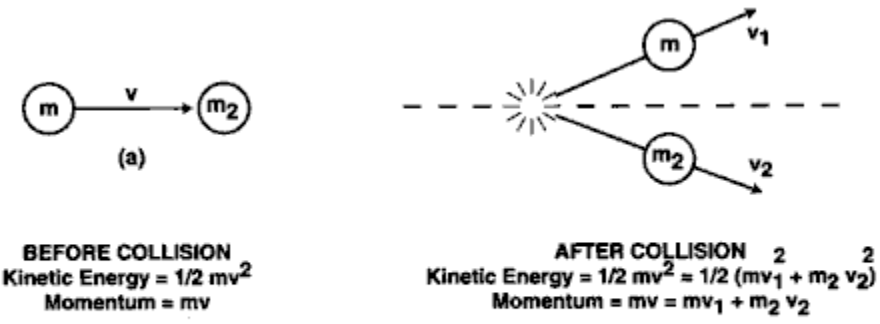


Figure 2-XVIII. An Elastic Collision

a. Because the energy of a neutron is degraded by interactions with atomic nuclei, the probability of a neutron interacting with a target material does not depend on electron density as it does for other types of radiation. For fast neutrons, the probability of collision is practically the same for all nuclei. Since, in accordance with the laws of mechanics, considerably more energy is transferred in a collision between two objects of similar mass, neutrons will transfer more energy to target nuclei of substances of low atomic mass rather than to heavy nuclei. The lightest element is common hydrogen, ^{1}H, the nucleus of which contains a single proton. The mass of a proton is essentially the same as that of a neutron; and so, when a neutron collides with a hydrogen nucleus it can lose a considerable fraction of its energy in the interaction. Therefore, substances containing large quantities of hydrogen are good neutron moderators.

b. The energy transfer mechanism with hydrogen accounts for more than 90% of the fast-neutron energy transfer up to 7 MeV occurring in wet, muscle-like tissue. The hydrogen nucleus then becomes ionized since it is accelerated away from its orbiting electron. An ion pair made up of the proton (H+) and the hydroxyl ion (OH-) is produced. The accelerated proton can, in turn, cause further secondary ionizations or excitations, spreading the damage of the original interaction. For neutrons with energies above 7 MeV, nuclear reactions with other tissue components become relatively important, but, even at 18 MeV, elastic scattering of protons constitutes about 70% of the energy absorbed in tissue.

224. Inelastic Collisions.

In inelastic collisions a neutron is captured by the target nucleus and a neutron of much lower energy is emitted leaving the target nucleus in an excited state. This energy is subsequently emitted in the form of electromagnetic radiation (Figure 2-XIX).

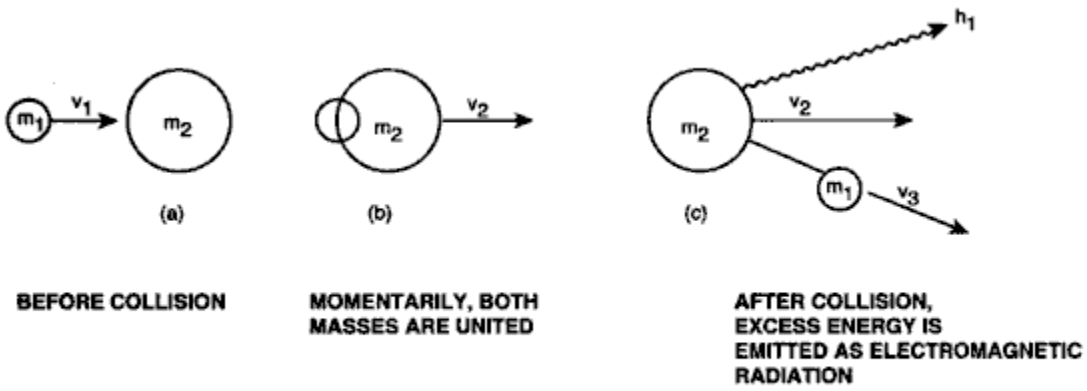


Figure 2-XIX. An Inelastic Collision

a. Thus, in inelastic collisions or capture the incident neutron energy is absorbed entirely. Much of it appears as subsequent electromagnetic radiation which is high penetrating and can interact with the target at some other location. (See discussion on photon interactions.)

b. Because the nucleus of an atom occupies so small a fraction of the total atomic volume, the probability of either elastic or inelastic scattering along a given neutron's path is very small. Even so, the energy transfer from an inelastic collision is significantly greater than that seen in the ionization or excitation interactions of gamma photons in tissue, and the penetrating ability of a fission-energy neutron is significantly less than that of gamma photons.

c. A wide variety of interactions of neutrons with elements present in tissues is possible. Incident neutrons of relatively low energy interact with and are captured by the nuclei of tissue elements, which become activated and emit a photon (gamma ray), which can penetrate over considerable distances prior to electron interactions. In interactions initiated by higher energy neutrons, the reaction products may have various energies depending on the energy level involved. Various biological materials have been used or suggested for measurements in connections with accidental exposure to neutrons. ^{32}P activity in body hair has been employed in the evaluation of exposures to neutrons. Both blood and whole body measurements of ^{24}Na activity are also important in the more accurate assessment of absorbed dose.

225. Neutron-to-Gamma Ratios.

a. The total dose due to initial radiation from a nuclear weapon can be divided into two components, neutrons and gamma rays. The neutron-to-gamma ratio is the ratio of neutron dose to gamma dose present at a specified point. The neutron-to-gamma ratio for a given total dose level is dependent on weapon yield and design, air density, and height-of-burst (HOB). Some typical neutron-to-gamma ratio values for 2600 centiGray (cGy) total dose to an unprotected individual are shown in Table 2-III.

*Table 2-III. Typical Neutron-to-Gamma and Neutron Dose-to-Total Dose Ratios**

Yield (Kt)	n/g	n/n + g	Range
0.1	4.6	0.82	360 meters
1.0	3.0	0.75	650 meters
10.0	1.6	0.62	1040 meters
100.0	0.47	0.32	1500 meters
1000.0	0.042	0.04	2280 meters

* Assumptions: HOB = $60W^{1/3}$ meters, where W = yield in kilotons; air density is equal to 0.9, relative to sea level; fission only device; total dose is equal to 2600 cGy.

b. As a general rule, the neutron-to-gamma ratio decreases with the range from the weapon's ground zero. This is due to the neutrons interacting with the air, creating secondary gamma. As a result, the gamma component decreases at a slower rate than does the neutron component. Thus, the ratios would be lower than the above values for a given yield at the 50-150 cGy dose levels because of the increased distance. These dose levels are typical of safety criteria. The ratios for vehicles and shelters depend on the specific neutron and gamma protection factors associated with the vehicle or shelter. These factors are based on the material used in construction. There are no typical ratios for vehicles, since each component of the ratio is effected differently by the associated radiation protection factor. However, for a tank, the protection factors are about 2 and 10 for neutrons and gammas, respectively. In other words, the neutron component would be decreased by a factor of two. Therefore, at least for tanks, the gamma radiation is more effectively stopped. This will significantly effect the neutron-to-gamma ratio within the vehicle. In this case, the neutron-to-gamma ratio would increase.

CHAPTER 3

EFFECTS OF NUCLEAR EXPLOSIONS

SECTION I - GENERAL

301. Introduction.

The basic differences in the mechanisms of energy production and related characteristics of conventional as compared with nuclear detonations were discussed in Chapter 2. In this chapter that discussion will be extended to consider the forms in which the energy produced in such detonations affects the surrounding environment. The location of the point of detonation in the environment is as important as the yield in determining the way the energy is distributed, and this factor will be discussed in some detail.

302. General Effects of Nuclear Explosions.

a. While the destructive action of conventional explosions is due almost entirely to the transmission of energy in the form of a blast wave with resultant mechanical damage, the energy of a nuclear explosion is transferred to the surrounding medium in three distinct forms: blast; thermal radiation; and nuclear radiation. The distribution of energy among these three forms will depend on the yield of the weapon, the location of the burst, and the characteristics of the environment. For a low altitude atmospheric detonation of a moderate sized weapon in the kiloton range, the energy is distributed roughly as follows:

(1) 50% as blast;

(2) 35% as thermal radiation; made up of a wide range of the electromagnetic spectrum, including infrared, visible, and ultraviolet light and some soft x-ray emitted at the time of the explosion; and

(3) 15% as nuclear radiation; including 5% as initial ionizing radiation consisting chiefly of neutrons and gamma rays emitted within the first minute after detonation, and 10% as residual nuclear radiation. Residual nuclear radiation is the hazard in fallout.

b. Considerable variation from this distribution will occur with changes in yield or location of the detonation. This is best shown by comparing the ranges of damage due to these effects of weapons of different size yields (Table 3-I).

*Table 3-I. Radii of Effects of Nuclear Weapons**

Effect	1 Kt	10 Kt	100 Kt	1000 Kt
Ionizing radiation (50% immediate transient ineffectivenss)	600m	950m	1400m	2900m
Ionizing radiation (50% latent lethality)	800m	110m	1600m	3200m
Blast (50% casualties)	140m	360m	860m	3100m
Thermal radiation (50% casualties, second degree burns under fatigue uniform)	369m	110m	3190m	8020m

* HOB 60W $^{1/3}$

c. The distribution of weapon energy yield is altered significantly by the enhanced radiation nuclear warhead. In simplest terms an enhanced radiation warhead is designed specifically to reduce the percentage of energy that is dissipated as blast and heat with a consequent increase in the percentage yield of initial radiation. Approximate percentage energies are 30% blast; 20% thermal; 45% initial radiation; and 5% residual radiation.

303. Initial Energy Transfer and Formation of Fireball.

a. Because of the tremendous amounts of energy liberated per unit mass in a nuclear detonation, temperatures of several tens of million degrees centigrade develop in the immediate area of the detonation. This is in marked contrast to the few thousand degrees of a conventional explosion. At these very high temperatures the nonfissioned parts of the nuclear weapon are vaporized. The atoms do not release the energy as kinetic energy but release it in the form of large amounts of electromagnetic radiation. In an atmospheric detonation, this electromagnetic radiation, consisting chiefly of soft x-ray, is absorbed within a few meters of the point of detonation by the surrounding atmosphere, heating it to extremely high temperatures and forming a brilliantly hot sphere of air and gaseous weapon residues, the so-called fireball. Immediately upon formation, the fireball begins to grow rapidly and rise like a hot air balloon. Within a millisecond after detonation, the diameter of the fireball from a 1 megaton (Mt) air burst is 150 m. This increases to a maximum of 2200 m within 10 seconds, at which time the fireball is also rising at the rate of 100 m/sec. The initial rapid expansion of

the fireball severely compresses the surrounding atmosphere, producing a powerful blast wave, discussed below.

b. The fireball itself emits enormous amounts of electromagnetic radiation, similar in its spectrum to sunlight. This is usually termed thermal radiation. The visible light component accounts for the blinding flash seen upon detonation as well as the subsequent brightness of the fireball, while the infrared component results in widespread burns and incendiary effects.

c. As it expands toward its maximum diameter, the fireball cools, and after about a minute its temperature has decreased to such an extent that it no longer emits significant amounts of thermal radiation. The combination of the upward movement and the cooling of the fireball gives rise to the formation of the characteristic mushroom-shaped cloud. As the fireball cools, the vaporized materials in it condense to form a cloud of solid particles. Following an air burst, condensed droplets of water give it a typical white cloudlike appearance. In the case of a surface burst, this cloud will also contain large quantities of dirt and other debris which are vaporized when the fireball touches the earth's surface or are sucked up by the strong updrafts afterwards, giving the cloud a dirty brown appearance. The dirt and debris become contaminated with the radioisotopes generated by the explosion or activated by neutron radiation and fall to earth as fallout.

d. The cloud rises for a period of approximately 10 minutes to a stabilized height which depends on the thermal output of the weapon and atmospheric conditions. It will continue to grow laterally assuming the familiar mushroom shape and may remain visible for an hour or more under favorable conditions. For example, the nuclear cloud from a 1 Mt surface burst will stabilize at an altitude of over 20 kilometers (km) and will have a mean lateral diameter of 35 km.

304. Types of Bursts.

The relative effects of blast, heat, and nuclear radiation will largely be determined by the altitude at which the weapon is detonated. Nuclear explosions are generally classified as air bursts, surface bursts, subsurface bursts, or high altitude bursts.

a. *Air Bursts.* An air burst is an explosion in which a weapon is detonated in air at an altitude below 30 km but at sufficient height that the fireball does not contact the surface of the earth. After such a burst, blast may cause considerable damage and injury. The altitude of an air burst can be varied to obtain maximum blast effects, maximum thermal effects, desired radiation effects, or a balanced combination of these effects. Burns to exposed skin may be produced over many square kilometers and eye injuries over a still larger area. Initial nuclear radiation will be a significant

hazard with smaller weapons, but the fallout hazard can be ignored as there is essentially no local fallout from an air burst. The fission products are generally dispersed over a large area of the globe unless there is local rainfall resulting in localized fallout. In the vicinity of ground zero, there may be a small area of neutron-induced activity which could be hazardous to troops required to pass through the area. Tactically, air bursts are the most likely to be used against ground forces.

b. *Surface Burst.* A surface burst is an explosion in which a weapon is detonated on or slightly above the surface of the earth so that the fireball actually touches the land or water surface. Under these conditions, the area affected by blast, thermal radiation, and initial nuclear radiation will be less extensive than for an air burst of similar yield, except in the region of ground zero where destruction is concentrated. In contrast with air bursts, local fallout can be a hazard over a much larger downwind area than that which is affected by blast and thermal radiation.

c. *Subsurface Burst.* A subsurface burst is an explosion in which the point of the detonation is beneath the surface of land or water. Cratering will generally result from an underground burst, just as for a surface burst. If the burst does not penetrate the surface, the only other hazard will be from ground or water shock. If the burst is shallow enough to penetrate the surface, blast, thermal, and initial nuclear radiation effects will be present, but will be less than for a surface burst of comparable yield. Local fallout will be very heavy if penetration occurs.

d. *High Altitude Burst.* A high altitude burst is one in which the weapon is exploded at such an altitude (above 30 km) that initial soft x-rays generated by the detonation dissipate energy as heat in a much larger volume of air molecules. There the fireball is much larger and expands much more rapidly. The ionizing radiation from the high altitude burst can travel for hundreds of miles before being absorbed. Significant ionization of the upper atmosphere (ionosphere) can occur. Severe disruption in communications can occur following high altitude bursts. They also lead to generation of an intense electromagnetic pulse (EMP) which can significantly degrade performance of or destroy sophisticated electronic equipment. There are no known biological effects of EMP; however, indirect effects may result from failure of critical medical equipment.

SECTION II - BLAST

305. Formation of Blast Wave.

a. As a result of the very high temperatures and pressures at the point of detonation, the hot gaseous residues move outward radially from the center of the explosion with very high velocities. Most of this material is contained within a relatively thin, dense

shell known as the hydrodynamic front. Acting much like a piston that pushes against and compresses the surrounding medium, the front transfers energy to the atmosphere by impulse and generates a steep-fronted, spherically expanding blast or shock wave. At first, this shock wave lags behind the surface of the developing fireball. However, within a fraction of a second after detonation, the rate of expansion of the fireball decreases to such an extent that the shock catches up with and then begins to move ahead of the fireball. For a fraction of a second, the dense shock front will obscure the fireball, accounting for the characteristic double peak of light seen with a nuclear detonation.

b. As it expands, the peak pressures of the blast wave diminish and the speed of propagation decreases from the initial supersonic velocity to that of sound in the transmitting medium. However, upon reflection from the earth's surface, the pressure in the wave will be reinforced by the fusion of the incident and the reflected wave (the Mach effect) described below.

c. A large part of the destruction caused by a nuclear explosion is due to blast effects. Objects within the path of the blast wave are subjected to severe, sharp increases in atmospheric pressure and to extraordinarily severe transient winds. Most buildings, with the exception of reinforced or blast- resistant structures, will suffer moderate to severe damage when subjected to overpressures of only 35.5 kiloPascals (kPa) (0.35 Atm). The velocity of the accompanying blast wind may exceed several hundred km/hr. Most materiel targets are drag- or wind-sensitive.

d. The range for blast effects increases significantly with the explosive yield of the weapon. In a typical air burst, these values of overpressure and wind velocity noted above will prevail at a range of 0.7 km for 1 kiloton (Kt) yield; 3.2 km for 100Kt; and 15.0 km for 10 Mt.

306. Propagation of Blast Wave in Air.

During the time the blast wave is passing through the superheated atmosphere in the fireball, it travels at supersonic velocities. After it leaves the vicinity of the fireball, it slows down to the normal speed of sound in the atmosphere. As long as the blast wave is expanding radially, its intensity decreases approximately as the square of the distance. When the expanding blast wave from a nuclear air burst strikes the surface of the earth, however, it is reflected (Figure 3-I), and the reflected wave reinforces and intensifies the primary wave.

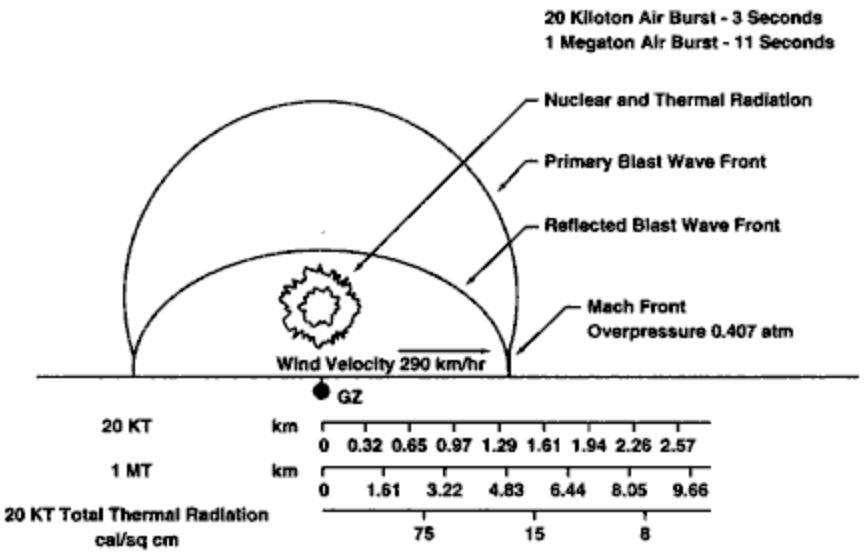


Figure 3-I. Chronological Development of an Air Burst

a. Targets in the vicinity of ground zero may actually be subjected to two blast waves: the initial or incident wave, followed slightly later by a secondary reflected wave. This limited region close to ground zero in which the incident and reflected waves are separate is known as the region of regular reflection.

b. Beyond the area of regular reflection as it travels through air which is already heated and compressed by the incident blast wave, the reflected wave will move much more rapidly and will very quickly catch up with the incident wave. The two then fuse to form a combined wave front known as the Mach stem. The height of the Mach stem increases as the blast wave moves outward and becomes a nearly vertical blast front. As a result, blast pressures on the surface will not decrease as the square of the distance, and most direct blast damage will be horizontally directed, e.g., on the walls of a building rather than on the roof.

c. As the height of burst for an explosion of given yield is decreased, or as the yield of the explosion for a given height of burst is increased, Mach reflection commences nearer to ground zero and the overpressure near ground zero becomes larger. However, as the height of burst is decreased, the total area of coverage for blast effects is also markedly reduced. The choice of height of burst is largely dependent on the nature of the target. Relatively resistant targets require the concentrated blast of a low altitude or surface burst, while sensitive targets may be damaged by the less severe blast wave from an explosion at a higher altitude. In the latter case a larger area and, therefore, a larger number of targets can be damaged.

d. A surface burst results in the highest possible overpressures near ground zero. In such a burst, the shock front is hemispherical in form, and essentially all objects are subjected to a blast front similar to that in the Mach region described above. A subsurface burst produces the least air blast, since most of the energy is dissipated in the formation of a crater and the production of a ground shock wave.

307. Static Overpressure and Dynamic Pressure.

a. Two distinct though simultaneous phenomena are associated with the blast wave in air:

(1) Static overpressure, i.e., the sharp increases in pressure due to compression of the atmosphere. These pressures are those which are exerted by the dense wall of air that comprises the wave front. The magnitude of the overpressure at any given point is directly proportional to the density of the air in the wave.

(2) Dynamic pressures, i.e., drag forces exerted by the strong transient blast winds associated with the movement of air required to form the blast wave. These forces are termed dynamic because they tend to push, tumble, and tear apart objects and cause their violent displacement.

b. In general, the static overpressure rises very abruptly from normal atmospheric in the unaffected air in front of the blast wave to a sharp peak (Figure 3-II). It then decreases behind the front. As the blast wave moves out from ground zero, the peak overpressure of the front diminishes while the decay of overpressure behind the front becomes more gradual. After traveling a sufficient distance from the fireball, the pressure behind the front actually drops below normal atmospheric pressure, the so-called negative phase of the blast wave.

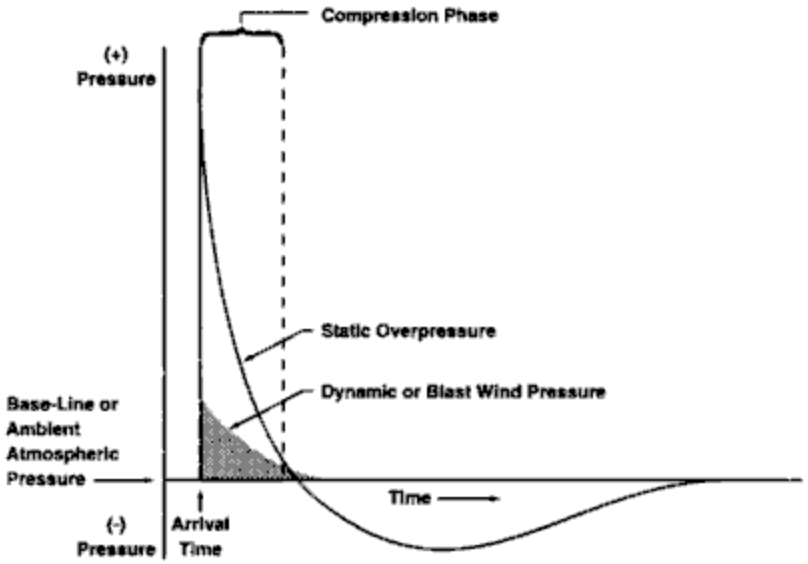


Figure 3-II. Variations of Overpressure and Dynamic Pressure with Time

c. In passing through the atmosphere, the blast wave imparts its energy to the molecules of the surrounding air, setting them into motion in the direction of the advancing shock front. The motion of these air molecules is manifested as severe transient winds, known as "blast winds," which accompany the blast wave. The destructive force associated with these winds is proportional to the square of their velocity and is measured in terms of dynamic pressure. These winds constitute decay forces which produce a large number of missiles and tumbling of objects. These dynamic forces are highly destructive.

d. Most of the material damage caused by a nuclear air burst is caused by a combination of the high static overpressures and the dynamic or blast wind pressures. The relatively long duration of the compression phase of the blast wave (Figure 3-II) is also significant in that structures weakened by the initial impact of the wave front are literally torn apart by the forces and pressures which follow. The compression and drag force phases together may last several seconds or longer, during which forces many times greater than those in the strongest hurricane are present. These persist even through the negative phase of a blast wave when a partial vacuum is present because of the violent displacement of air.

e. It is of practical value to examine the variation in pressure at a fixed location as a function of time. For a short period of time after a nuclear air burst, there will be no increase in pressure since it takes a finite time for the shock front to reach a given point. This arrival time, which may range from a few seconds to minutes, will depend

primarily on the distance of the location from the center of burst and to a lesser extent on the yield of the explosion. Initially, the speed of the shock front is many times the speed of sound because it is traveling through superheated air, but as it travels away from the fireball it slows down to the speed of sound, 330 m/sec, in normal atmospheres. With high yield detonations, the early velocity of the shock front and the distance traveled through superheated air is greater. Therefore, time is somewhat less. Upon arrival of the shock front, both the static overpressure and the dynamic pressure increase almost immediately from zero to their maximum values. The peak values of pressure will, of course, depend on the distance from ground zero, the height of burst, and the yield and will be further modified by differences in terrain and meteorological conditions. With passage of the blast front, both the static and dynamic pressures decay, though at slightly different rates. Most blast damage will be experienced during the positive or compression phase of the wave. The duration of this positive phase increases with yield and distance from ground zero and ranges from 0.2 to 0.5 sec for a 1 KT nuclear air burst to 4 to 10 sec for a 10 Mt explosion. This compares with only a few hundredths of a second for the duration of a blast wave from a conventional high-explosive detonation.

f. Because of the much longer duration of the blast wave from a nuclear explosion, structures are subjected to maximum loading for correspondingly longer periods of time, and damage will be much more extensive for a given peak overpressure than might otherwise be expected. During the negative phase, which is generally of even longer duration, the static pressure will drop below normal atmospheric pressure and the blast winds will actually reverse direction and blow back towards ground zero. Damage sustained during the negative phase is generally minor, however, because the peak values of underpressure and wind velocity are relatively low. Blast effects associated with positive and negative phase pressures are shown in Figure 3-III.

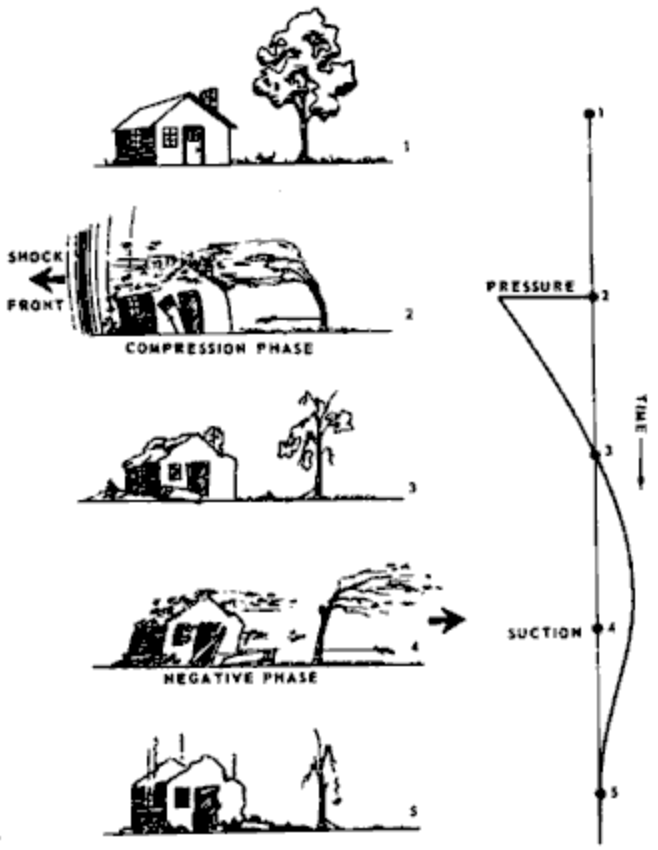


Figure 3-III. Variations of Blast Effects Associated with Positive and Negative Phase Pressures with Time

308. Blast Loading.

When a blast wave strikes the surface of a hard target, such as a building, the reflected wave will reinforce the incident wave, and the face of the building will be subjected to overpressures 2 to 8 times that of the incident wave alone. The severity of this additional stress depends on many factors, including the peak overpressure of the incident blast wave, as well as the angle at which the wave strikes the building. As the shock front advances, it bends or diffracts around the building, and the pressure on the front wall decreases rapidly. However, during the brief interval in which the blast wave has not yet engulfed the entire structure, a considerable pressure gradient exists from front to rear that places a severe stress on the building. For small objects, this period of so-called diffraction loading is so small that no significant stress is encountered. For large buildings, however, the stress of diffraction loading will be considerable. Even after the shock front has passed across the building, the structure will still be subjected to a severe compression force and to severe drag forces from the

transient winds. The actual overpressures required to produce severe damage to diffraction sensitive targets are actually quite low. Table 3-II depicts failure of sensitive structural elements when exposed to overpressure blast loading.

Table 3-II. Failure of Overpressure Sensitive Structural Elements

Structural element	Failure	Approximate side-on peak overpressure (kPa)	Approximate slant range (km) 20 Kt	1 Kt
Glass windows, large and small	Shattering usually, occasional frame failure	3.45 - 6.9	6-10	20-30
Corrugated asbestos siding	Shattering	6.9 - 13.8	3-6	12-22
Corrugated steel or aluminum paneling	Connection failure followed by buckling	6.9 - 13.8	3-6	12-22
Brick wall panel, 20 cm or cm thick (not reinforced)	Shearing and flexture failures	20.7 - 69.0	1-3	4-10
Wood siding panels standard house construction	Usually failure occurs at the main connections, allowing a whole panel to be blown in	6.9 - 13.8	3-6	12-22
Concrete or cinder-block wall panels, 28 cm or 30 cm thick (not reinforced)	Shattering of the wall	10.35 - 38.0	1.5-4	6.5-15

309. Drag Loading.

All objects in the path of the blast wave, regardless of size or structure, will be subject to the dynamic pressure loading or drag forces of the blast winds. Drag loading is influenced to a moderate degree by the shape of the target. Round objects are relatively unaffected by the winds, while flat or recessed surfaces offer great resistance and hence are subjected to increased impact pressure and probability of damage. The effect of dynamic pressure is generally dependent on the peak value of dynamic pressure and its duration. While the dynamic pressure at the face of a building is generally less than the peak overpressure due to the blast wave and its reflection, the period of dynamic loading is much longer than that of diffraction

loading, and hence the damage to frame-type buildings, bridges, and other structures will be considerable. Equipment and personnel are relatively resistant to static overpressures but highly vulnerable to dynamic pressure. For example, military vehicles, from jeeps to tanks, are most likely to suffer damage when pushed, overturned, and thrown about by the blast winds. Likewise, blast winds are the cause of most blast injuries. Because of the violence of the winds associated with even low values of overpressure, mechanical injuries due to missiles sent into motion by the winds or to violent bodily translation will far outnumber direct blast injuries due to actual compression of the organism.

310. Shock Waves in Other Media.

a. In surface and subsurface bursts, a sizable portion of the yield is transmitted in the form of ground or water shock waves. In the case of a surface burst on land, a crater is formed at ground zero, the size of which depends primarily upon yield. Relatively little damage beyond a distance of approximately three crater radii will occur due to ground shock. Most damage will be due to the accompanying air blast wave. In subsurface bursts the crater will be formed either by ejection of material as in a shallow explosion or by the collapse of ground into the cavity formed by a deeper explosion. Since the overpressure in a ground shock wave decreases very rapidly with distance, shock damage will again be confined to a region close to the point of detonation.

b. Ground shock waves will also be induced as a result of an air burst. If the overpressure in the blast wave is very large, the ground shock will penetrate some distance into the ground and may damage underground structures and buried utilities, etc.

c. Because of the density and relative incompressibility of water, shock waves in that medium have very high peak overpressures and velocities of propagation. The peak overpressure at a distance of 1 km from a 10 Kt underwater burst is approximately 6080 kPa (60 atm (atmospheres of pressure)), while the peak overpressure in air at the same distance from an air burst is only 111.4 kPa (1.1 atm). The resulting surface waves at this distance will be approximately 10 m in height. The shock front will also travel at approximately five times the speed of the blast wave in air. Severe damage to naval vessels may result from the shock wave produced by an underwater or water surface burst. Although the major portion of the shock energy is propagated in the water, a significant amount is also transferred through the surface as a typical air blast. This blast wave could probably be the principal source of damage to land targets if the explosion occurred in a coastal area.

SECTION III - THERMAL RADIATION

311. Formation of Thermal Radiation.

Large amounts of electromagnetic radiation in the visible, infrared, and ultraviolet regions of the electromagnetic spectrum are emitted from the surface of the fireball within the first minute or less after detonation. This thermal radiation travels outward from the fireball at the speed of light, 300,000 km/sec. The chief hazard of thermal radiation is the production of burns and eye injuries in exposed personnel. Such thermal injuries may occur even at distances where blast and initial nuclear radiation effects are minimal. Absorption of thermal radiation will also cause the ignition of combustible materials and may lead to fires which then spread rapidly among the debris left by the blast. The range of thermal effects increases markedly with weapon yield.

312. Propagation of Thermal Energy.

a. Most of the energy released in the fission or fusion processes is initially in the form of the kinetic energy of the products of the reactions (e.g., fission fragments, etc.). Within millionths of a second after detonation, numerous inelastic collisions of these vaporized atoms give rise to a plasma of intensely hot weapon residues. Since the temperature of this system is of several tens of million degrees centigrade, it emits enormous quantities of energy in the form of electromagnetic radiation. This radiation is subsequently absorbed by the surrounding atmosphere, which is heated to extremely high temperatures, causing it to emit additional radiation of slightly lower energy. This complex process of radiative transfer of energy is the basic mechanism by which the fireball is formed and expands.

b. Because this thermal radiation travels at the speed of light, and its mean free path (distance between point of emission and point of absorption) is relatively long, the initial expansion of the fireball is extremely rapid, much more so than the outward motion of gaseous material from the center of the burst responsible for production of the blast wave. Consequently, the blast wave front at first lags behind the radiative front (surface of the fireball).

c. However, as the fireball expands and its energy is deposited in an ever-increasing volume its temperature decreases and the transfer of energy by thermal radiation becomes less rapid. At this point, the blast wave front begins to catch up with the surface of the fireball and then moves ahead of it, a process called hydrodynamic separation. Due to the tremendous compression of the atmosphere by the blast wave, the air in front of the fireball is heated to incandescence. Thus, after hydrodynamic separation, the fireball actually consists of two concentric regions: the hot inner core known as the isothermal sphere; and an outer layer of luminous shock-heated air.

d. The outer layer initially absorbs much of the radiation from theisothermal sphere and hence the apparent surface temperature of the fireball and the amount of radiation emitted from it decreases after separation. But, as the shock front advances still farther, the temperature of the shocked air diminishes and it becomes increasingly transparent. This results in an unmasking of the still incandescent isothermal region and an increase in the apparent surface temperature of the fireball. This phenomena is referred to as breakaway.

313. Rate of Thermal Emission.

a. The rate of thermal emission from the fireball is governed by its apparent surface temperature. From the foregoing discussion, it should be apparent that the thermal output of a nuclear air burst will then occur in two pulses (Figure 3-IV), an initial pulse, consisting primarily of ultraviolet radiation, which contains only about 1% of the total radiant energy of the explosion and is terminated as the shock front moves ahead of the fireball, and a second pulse which occurs after breakaway.

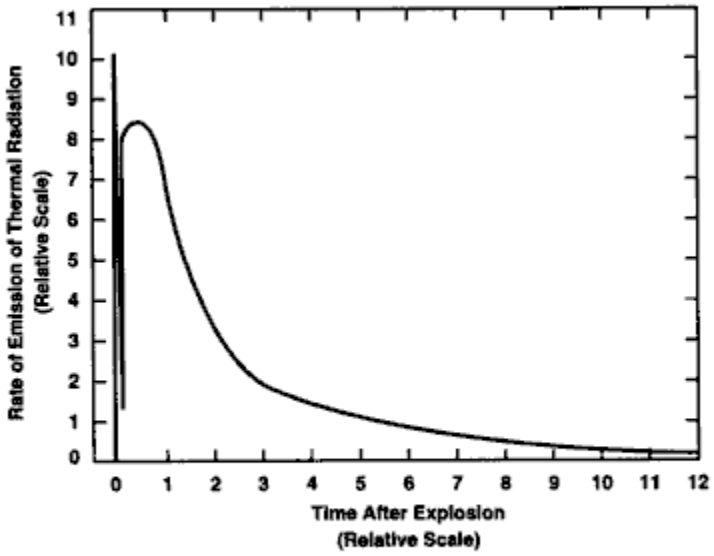


Figure 3-IV. Emission of Thermal Radiation in Two Pulses in an Air Blast

b. The thermal radiation emitted from the fireball surface during the second thermal pulse is responsible for most of the thermal effects. It consists chiefly of radiation in the infrared, visible, and ultraviolet regions of the electromagnetic spectrum. Thermal exposure (measured in joules per unit area of exposed surface) will be less farther from the center of the explosion because the radiation is spread over a greater area and is attenuated in passing through the intervening air. Since the fireball is very close to a point source of thermal radiation, the quantity of thermal radiation at any given point varies approximately with the square of the distance from the explosion. The inverse

square law does not apply exactly because thermal radiation, particularly ultraviolet, will also be absorbed and scattered by the atmosphere. The degree of atmospheric visibility affects the attenuation of thermal energy with distance to a limited degree, but less than would be expected from the purely absorptive properties of the atmosphere, because the decrease in transmission is largely compensated by an increase in scattered radiation.

314. Shielding.

Since thermal radiation travels in straight lines from the fireball (unless scattered) any opaque object interposed between the fireball and the target will act as a shield and provide significant protection from thermal radiation. If a significant amount of scattering is present, as is the case when visibility is poor, thermal radiation will be received from all directions and shielding will be less effective.

315. Yield and Altitude.

a. *Yield*. The total amount of thermal radiation, the period of time during which it is emitted, and the range for thermal effects increase with the yield of the nuclear explosion (Figure 3-V).

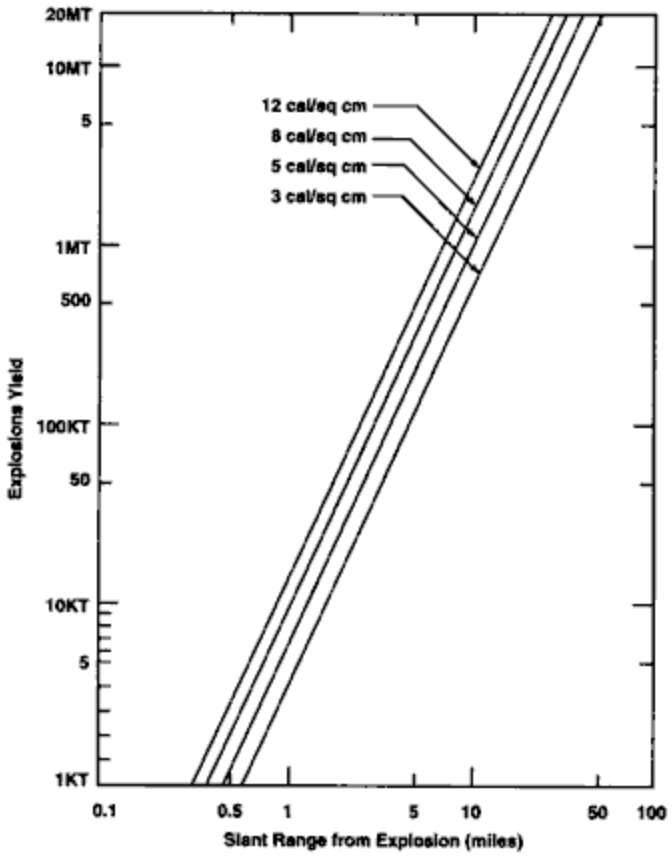


Figure 3-V. Slant Ranges for Specified Radient Exposures as Functions of Energy Yield of the Explosion

b. *Altitude Effects*. The thermal radiation intensity at a given point will depend on the altitude and the type of burst. In general, the thermal hazard is greatest in the case of a low altitude air burst. General thermal effects will be less for surface bursts and frequently nonexistent for subsurface bursts. In surface bursts a large part of the thermal energy is absorbed by the ground or water around ground zero. In addition, shielding due to terrain irregularities of dust, moisture, and various gases in the air near the surface of the earth will tend to reduce the amount of thermal energy reaching a target. In subsurface bursts without appreciable penetration, most of the thermal energy is absorbed and dissipated in heating and vaporizing soil and water below the surface.

c. *High Altitude Effects*. In high altitude air bursts (above 30 km), the low density of the atmosphere alters the nature of the thermal radiation process because the primary thermal radiation is absorbed in a much larger volume of air, and the temperature of the system is correspondingly less. While a greater percentage of the yield of the explosion appears in the form of thermal radiation, much of the radiation is emitted so

slowly that it is ineffective. About 25-35% of the total yield is emitted in a single pulse of very short duration. Moreover, because of the relatively great distance between the center of the burst and the earth's surface, the intensity of thermal radiation at ground level is generally low.

316. Thermal Effects.

a. When thermal radiation strikes an object, part will be reflected, part will be transmitted, and the rest will be absorbed. The fraction of the incident radiation that is absorbed depends on the nature and color of the material. A thin material may transmit a large part of the radiant energy striking it. A light colored object may reflect much of the incident radiation and thus escape damage. Thermal damage and injury is due to the absorption of large amounts of thermal energy within relatively short periods of time. The absorbed thermal radiation raises the temperature of the absorbing surface and results in scorching, charring, and possible ignition of combustible organic materials, such as wood, paper, fabrics, etc. If the target material is a poor thermal conductor, the absorbed energy is largely confined to a superficial layer of the material.

b. The radiation exposure (# Joules/sq/cm) required for the ignition of materials and other thermal effects increases with the yield of the weapon (Table 3-III). This is so because increased thermal energy is required to compensate for energy lost via conduction and convection during the longer thermal pulse of higher yield weapons. For lower yield weapons, the thermal pulse is so short that there is not much time for these processes to cool the exposed surface. Hence, a much higher percentage of the deposited thermal energy is effective in producing thermal damage. This increased thermal requirement does not mean that the thermal hazard is less significant for higher yields. On the contrary, the total thermal energy released during a nuclear explosion increases markedly with yield, and the effects extend over much greater distances. Therefore, although more thermal energy is required to produce a given thermal response for a large yield explosion, the effective range to which this level extends is very much greater.

*Table 3-III. Approximate Radient Exposures for Ignition of Fabrics for Low Air Burst**

Material	Wt (g/m^2)	Color	Effect on material	Ignition Exposure 35 kilotons ground Joules sq cm	35 kilotons ground Range** (km)	20 megatons ground Joules sq cm	20 megatons ground Range** (km)
a. Clothing Fabrics:							
Cotton	298	White	Ignites	134	2.1	355	20.9
		Khaki	Tears on flexing	71	2.7	142	33.2
		Khaki	Ignites	84	2.5	163	30.9
		Olive	Tears on flexing	38	3.4	88	42.1
		Olive	Ignites	58	2.9	88	42.1
		Dk Blue	Tears on flexing	46	3.2	71	46.9
		Dk Blue	Ignites	58	2.9	88	42.1
Cotton corduroy	298	Brown	Ignites	46	3.2	92	41.2
Cotton denim, new	372	Blue	Ignites	50	3.1	184	29.1
Cotton shirting, new	112	Khaki	Ignites	58	2.9	117	36.5
Cotton-nylon mixture	186	Olive	Tears on flexing	33	3.6	71	46.9
		Olive	Ignites	50	3.1	222	26.5
Wool	298	White	Tears on flexing	58	2.9	109	37.9
		Khaki	Tears on flexing	58	2.9	142	33.2
		Olive	Tears on flexing	38	3.4	79	44.5
		Dk Blue	Tears on flexing	33	3.6	75	45.7
	744	Dk Blue	Tears on flexing	58	2.9	109	37.9
Rainware (double neoprene-coated)	335	Olive	Begins to melt	21	4.4	54	53.8
Nylon twill		Olive	Tears on flexing	33	3.6	92	41.2
b. Drapery Fabrics:							
Rayon gabardine	223	Black	Ignites	38	3.4	109	37.9
Rayon-acetate	186	Wine	Ignites	38	3.4	117	36.5
Rayon gabardine	260	Gold	Ignites	***		#117	36.5
Rayon twill lining	112	Black	Ignites	29	3.8	104	38.8
Rayon twill lining	112	Beige	Ignites	54	3.0	117	36.5
Acetate-sheeting	112	Black	Ignites	#42	3.3	#146	32.7
Cotton heavy drapes	484	Dark	Ignites	63	2.8	142	33.2
c. Tent Fabrics:							
Canvas (cotton)	446	White	Ignites	54	3.0	213	27.1
Canvas	446	O. Drab	Ignites	50	3.1	117	36.5
d. Other Fabrics:							
Cotton chenille bedspread		Lt Blue	Ignites	***		63	49.8
Cotton venetian blind tape, dirty		White	Ignites	42	3.3	92	41.2
Cotton venetian blind tape		White	Ignites	#54	3.0	#130	34.7
Cotton muslin window shade	298	Green	Ignites	29	3.8	79	44.5

* Radient exposures for indicated responses (except where marked#) are estimated valid to +25% under standard laboratory conditions. Under typical field conditions, values are estimated within +50% with a greater likelihood of the higher than lower values. For materials marked #, ignition levels are estimated to be valid within +50% under laboratory conditions and within 100% under field conditions.

** Ground ranges calculated for good visibility conditions.

*** Data not available or appropriate scaling not known.

c. Actual ignition of materials exposed to thermal radiation is highly dependent on the width of the thermal pulse (which is dependent on weapon yield) and the nature of the material, particularly its thickness and moisture content. At locations close to ground zero where the radiant thermal exposure exceeds 125 Joules/sq cm, almost all ignitable materials will flame, although burning may not be sustained (Table 3-III). On the other hand, at greater distances only the most easily ignited materials will flame, although charring of exposed surfaces may occur. The probability of

significant fires following a nuclear explosion depends on the density of ignition points, the availability and condition of combustible material (whether hot, dry, wet), wind, humidity, and the character of the surrounding area. Incendiary effects are compounded by secondary fires started by the blast wave effects such as from upset stoves and furnaces, broken gas lines, etc. In Hiroshima, a tremendous fire storm developed within 20 minutes after detonation. A fire storm burns in upon itself with great ferocity and is characterized by gale force winds blowing in towards the center of the fire from all points of the compass. It is not, however, a phenomenon peculiar to nuclear explosions, having been observed frequently in large forest fires and following incendiary raids during World War II.

SECTION IV - NUCLEAR RADIATION

317. Sources of Nuclear Radiation.

Blast and thermal effects occur to some extent in all types of explosions, whether conventional or nuclear. The release of ionizing radiation, however, is a phenomenon unique to nuclear explosions and is an additional casualty producing mechanism superimposed on blast and thermal effects. This radiation is basically of two kinds, electromagnetic and particulate, and is emitted not only at the time of detonation (initial radiation) but also for long periods of time afterward (residual radiation). Initial or prompt nuclear radiation is that ionizing radiation emitted within the first minute after detonation and results almost entirely from the nuclear processes occurring at detonation. Residual radiation is defined as that radiation which is emitted later than 1 minute after detonation and arises principally from the decay of radioisotopes produced during the explosion.

318. Initial Radiation.

About 5% of the energy released in a nuclear air burst is transmitted in the form of initial neutron and gamma radiation. The neutrons result almost exclusively from the energy producing fission and fusion reactions, while the initial gamma radiation includes that arising from these reactions as well as that resulting from the decay of short-lived fission products. The intensity of initial nuclear radiation decreases rapidly with distance from the point of burst due to the spread of radiation over a larger area as it travels away from the explosion, and to absorption, scattering, and capture by the atmosphere. The character of the radiation received at a given location also varies with distance from the explosion. Near the point of the explosion, the neutron intensity is greater than the gamma intensity, but with increasing distance the neutron-gamma ratio decreases. Ultimately, the neutron component of initial radiation becomes negligible in comparison with the gamma component. The range for significant levels of initial radiation does not increase markedly with weapon yield

and, as a result, the initial radiation becomes less of a hazard with increasing yield. With larger weapons, above 50 Kt, blast and thermal effects are so much greater in importance that prompt radiation effects can be ignored.

319. Residual Radiation.

The residual radiation hazard from a nuclear explosion is in the form of radioactive fallout and neutron-induced activity. Residual ionizing radiation arises from:

a. *Fission Products*. These are intermediate weight isotopes which are formed when a heavy uranium or plutonium nucleus is split in a fission reaction. There are over 300 different fission products that may result from a fission reaction. Many of these are radioactive with widely differing half-lives. Some are very short, i.e., fractions of a second, while a few are long enough that the materials can be a hazard for months or years. Their principal mode of decay is by the emission of beta and gamma radiation. Approximately 60 grams of fission products are formed per kiloton of yield. The estimated activity of this quantity of fission products 1 minute after detonation is equal to that of 1.1×10^{21} Bq (30 million kilograms of radium) in equilibrium with its decay products.

b. *Unfissioned Nuclear Material.* Nuclear weapons are relatively inefficient in their use of fissionable material, and much of the uranium and plutonium is dispersed by the explosion without undergoing fission. Such unfissioned nuclear material decays by the emission of alpha particles and is of relatively minor importance.

c. *Neutron-Induced Activity*. If atomic nuclei capture neutrons when exposed to a flux of neutron radiation, they will, as a rule, become radioactive (neutron-induced activity) and then decay by emission of beta and gamma radiation over an extended period of time. Neutrons emitted as part of the initial nuclear radiation will cause activation of the weapon residues. In addition, atoms of environmental material, such as soil, air, and water, may be activated, depending on their composition and distance from the burst. For example, a small area around ground zero may become hazardous as a result of exposure of the minerals in the soil to initial neutron radiation. This is due principally to neutron capture by sodium (Na), manganese, aluminum, and silicon in the soil. This is a negligible hazard because of the limited area involved.

320. Fallout.

a. *Worldwide Fallout.* After an air burst the fission products, unfissioned nuclear material, and weapon residues which have been vaporized by the heat of the fireball will condense into a fine suspension of very small particles 0.01 to 20 micrometers in diameter. These particles may be quickly drawn up into the stratosphere, particularly

so if the explosive yield exceeds 10 Kt. They will then be dispersed by atmospheric winds and will gradually settle to the earth's surface after weeks, months, and even years as worldwide fallout. The radiobiological hazard of worldwide fallout is essentially a long-term one due to the potential accumulation of long-lived radioisotopes, such as strontium-90 and cesium-137, in the body as a result of ingestion of foods which had incorporated these radioactive materials. This hazard is much less serious than those which are associated with local fallout and, therefore, is not discussed at length in this publication. Local fallout is of much greater immediate operational concern.

b. *Local Fallout.* In a land or water surface burst, large amounts of earth or water will be vaporized by the heat of the fireball and drawn up into the radioactive cloud. This material will become radioactive when it condenses with fission products and other radiocontaminants or has become neutron-activated. There will be large amounts of particles of less than 0.1 micrometer to several millimeters in diameter generated in a surface burst in addition to the very fine particles which contribute to worldwide fallout. The larger particles will not rise into the stratosphere and consequently will settle to earth within about 24 hours as local fallout. Severe local fallout contamination can extend far beyond the blast and thermal effects, particularly in the case of high yield surface detonations. Whenever individuals remain in a radiologically contaminated area, such contamination will lead to an immediate external radiation exposure as well as a possible later internal hazard due to inhalation and ingestion of radiocontaminants. In severe cases of fallout contamination, lethal doses of external radiation may be incurred if protective or evasive measures are not undertaken. In cases of water surface (and shallow underwater) bursts, the particles tend to be rather lighter and smaller and so produce less local fallout but will extend over a greater area. The particles contain mostly sea salts with some water; these can have a cloud seeding affect causing local rainout and areas of high local fallout. For subsurface bursts, there is an additional phenomenon present called "base surge." The base surge is a cloud that rolls outward from the bottom of the column produced by a subsurface explosion. For underwater bursts the visible surge is, in effect, a cloud of liquid (water) droplets with the property of flowing almost as if it were a homogeneous fluid. After the water evaporates, an invisible base surge of small radioactive particles may persist. For subsurface land bursts, the surge is made up of small solid particles, but it still behaves like a fluid. A soil earth medium favors base surge formation in an underground burst.

c. *Meteorological Effects.* Meteorological conditions will greatly influence fallout, particularly local fallout. Atmospheric winds are able to distribute fallout over large areas. For example, as a result of a surface burst of a 15 Mt thermonuclear device at Bikini Atoll on March 1, 1954, a roughly cigar-shaped area of the Pacific extending

over 500 km downwind and varying in width to a maximum of 100 km was severely contaminated. Snow and rain, especially if they come from considerable heights, will accelerate local fallout. Under special meteorological conditions, such as a local rain shower that originates above the radioactive cloud, limited areas of heavy contamination may be formed.

CHAPTER 4

BIOLOGICAL EFFECTS OF A NUCLEAR EXPLOSION

SECTION I - GENERAL

401. Introduction.

Chapter 3 described the physical characteristics of nuclear explosions. This chapter will consider the biological effects of blast and thermal radiation. The material to be presented is intended to supplement the material on the clinical aspects of blast and thermal injuries described in Chapter 6. The basic scientific aspects of radiation injury will be discussed in Chapter 5.

SECTION II - BLAST INJURY

402. General.

a. The basic physical effects of a blast wave are described in Chapter 3 along with how the wave is formed. There are two basic types of blast forces which occur simultaneously in a nuclear detonation blast wave. These are: direct blast wave overpressure forces, measured in terms of atmospheres of overpressure; and indirect blast wind drag forces, measured best in terms of the velocities of the wind which cause them. The most important blast effects, insofar as production of casualties requiring medical treatment is concerned, will be those due to the blast wind drag forces. Direct overpressure effects do not extend out as far from the point of detonation and are frequently masked by drag force effects as well as by thermal effects.

b. However, direct blast effects can contribute significantly to the immediate deaths and injuries sustained close to the point of detonation and, therefore, do constitute an important total casualty producing effect in the large area of lethal damage associated with a given nuclear detonation. Personnel in fortifications or heavy vehicles such as tanks who are protected from radiation and thermal and blast wind effects may be subjected to complex patterns of direct overpressures since blast waves can enter such structures and be reflected and reinforced within them.

403. Direct Blast Injury.

a. When a blast wave is incident upon a target, the nature and probability of damage will depend upon a number of variables in the characteristics of the blast wave and of the target. Important variables of the blast wave include: the rate of pressure rise at the

blast wave front, the magnitude of the peak overpressure, and the duration of the blast wave. Those of the target include: size, mass, density, resistance to deformity, etc. If the target is human, then additional factors such as age, physical condition, and the presence of disease or other injury become important.

b. When the blast wave acts directly upon a resilient target such as the human body, rapid compression and decompression result in transmission of pressure waves through the tissues. These waves can be quite severe and will result in damage primarily at junctions between tissues of different densities (bone and muscle) or at the interface between tissue and air spaces. Lung tissue and the gastrointestinal system, both of which contain air, are particularly susceptible to injury. The resulting tissue disruptions can lead to severe hemorrhage or to air embolism, either of which can be rapidly fatal. Perforation of the ear drums would be a common but a minor blast injury.

c. The range of overpressures associated with lethality can be quite variable. It has been estimated that overpressures as low as 193 kPa (1.9 atm) can be lethal, but that survival is possible with overpressures as high as 262 kPa (2.5 atm). Atypical range of probability of lethality with variation in overpressure is summarized in Table 4-I. These are rough estimates based on selected experimental data, and there will be some differences between these figures and tabulations based upon other experimental work. In addition these numbers apply only to unreinforced, unreflected blast waves. When blast waves are complicated by reinforcement and reflection, estimation or measurement of the overpressures associated with specific injuries becomes quite complex. The significant thing shown by the data in Table 4-I is that the human body is remarkably resistant to static overpressure, particularly when compared with rigid structures such as buildings. Shattering of an unreinforced cinder block panel, for example, will occur at 10.1-20.2 kPa (0.1-0.2 atm).

Table 4-I. Range of Lethality at Peak Overpressure

Lethality (approximate %)	Peak overpressure (k Pa)
1	160 - 230
50	230 - 400
100	400 +

d. Overpressures considerably lower than those listed in Table 4-I will cause injuries which are not lethal. Lung damage and eardrum rupture are two useful biomedical parameters to use as examples, since one is a relatively serious injury, usually

requiring hospitalization even if not lethal, while the other is a minor injury, often requiring no treatment at all.

(1) The threshold level of overpressure which is estimated to cause lung damage is about 68.9 kPa for a simple unreinforced, unreflected blast wave. There will be considerable variation in this value with differing conditions of exposure.

(2) The threshold value for eardrum rupture is probably around 22 kPa (0.2 atm) and that overpressure associated with a 50% probability of eardrum rupture ranges from 90 to 130 kPa (0.9 to 1.2 atm).

e. From this it can be seen that casualties requiring medical treatment from direct blast effects could theoretically be produced by overpressures greater than 70 kPa. However, direct blast injuries will not occur by themselves; and in general, other effects, such as indirect blast injuries and thermal injuries are so severe at the ranges associated with these overpressures that patients with direct blast injuries will comprise a very small part of the patient load.

404. Indirect Blast Wind Drag Forces.

a. *Blast Winds*. The drag forces of the blast winds are proportional to the velocities and duration times of those winds, which in turn vary with distance from the point of detonation, yield of the weapon, and altitude of the burst. These winds are relatively short in duration but are extremely severe. They can be much greater in velocity than the strongest hurricane winds and may reach several hundred kilometers per hour. Considerable injury can result, due either to missiles or to the physical displacement of human bodies against objects and structures in the environment.

b. *Probability of Indirect Blast Injury*. The distance from the point of detonation at which severe indirect injury will occur is considerably greater than that for equally serious direct blast injuries. It is difficult to give precise ranges at which these indirect injuries are likely to occur because of the marked effect of variations in the environment. However, that range at which the peak overpressure is about 20.3 kPa (0.2 atm) is a reasonable reference distance at which the probability of serious indirect injury is high. Injuries can occur at greater ranges, and casualties will be generated at greater ranges, but not consistently.

405. Missile Injury.

The probability of injury from a missile depends upon a number of factors.

a. *The Number of Missiles.* The number of missiles which can be generated by the blast winds depends to some extent upon the environment. Certain terrain, such as desert, is particularly susceptible to missile forming effects of winds. However, the drag forces of the blast winds produced by nuclear detonations are so great that almost any form of vegetation or structure will be broken apart or fragmented into a variety of missiles. As a result, large numbers and a great variety of missiles will be generated in almost any environment. Single missile injuries will be rare and multiple, varied missile injuries will be common. As a result, the overall severity and significance of missile injuries is greatly increased. Table 4-II gives an indication of the ranges out to which significant missile injuries would be expected.

*Table 4-II. Ranges for Different Probabilities of Injury from Small Missiles**

Yield (Kt)	Range for 1% probability of serious injury	Range for 50% probability of serious injury	Range for 99% probability of serious injury
1	0.28 km	0.22 km	0.17 km
10	0.73 km	0.57 km	0.44 km
20	0.98 km	0.76 km	0.58 km
50	1.4 km	1.1 km	0.84 km
100	1.9 km	1.5 km	1.1 km
200	2.5 km	1.9 km	1.5 km
500	3.6 km	2.7 km	2.1 km
1000	4.8 km	3.6 km	2.7 km

* Incidence of head injury based on incidence of perforation of skin and tissue. Missiles used were 10 gm in weight.

b. *The Kinetic Energy and Shape of the Missiles.* Several separate factors are involved here, but a detailed discussion of complex missile ballistics is beyond the scope of this handbook. The major factor in how missiles are accelerated depends upon the wind velocity and the size and weight of the missiles. The wind velocity is the maximum, since objects cannot be made to go faster than the winds themselves. Therefore, all these missiles will be low velocity in nature. None will be high velocity, such as is produced with small arms fire. The weight or mass of an object and the duration times of the winds determine whether or not that object will be accelerated maximally. Light objects will be accelerated rapidly up to the maximum possible velocity, whereas heavy objects may not be. The velocity is important because the probability of a penetrating injury increases with increasing velocity, particularly for small, sharp missiles such as glass fragments. Table 4-III shows typical experimental data for probability of penetration related to size and velocity of glass fragments of various weights. The table also lists the kinetic energy associated with each weight and velocity. The progression in energy is reversed, and it can be seen that heavier objects require higher kinetic energies to penetrate, at least in this particular experimental system. Heavy blunt missiles will not penetrate but can result in significant injury,

particularly fractures. For example, a velocity of about 4.6 meters/sec is a threshold velocity for skull fracture for a 4.5 kg missile.

*Table 4-III. Probability of Penetration of Glass Fragments and Associated Kinetic Energy Related to Size and Velocity**

Mass of glass fragments (grams)	1%	50%	99%
	Impact velocity (m/sec)**/Kinetic energy (joules)***		
0.1	78/0.3	136/0.9	243/3.0
0.5	53/0.7	91/2.1	161/6.5
1.0	46/1.1	82/3.4	143/10.2
10.0	38/7.2	60/18.0	118/70.0

* The penetrating injury example here is for the abdominal cavity.
** Impact velocity is in m/sec. Conversion to cm/sec is necessary to determine kinetic energy in joules.
*** Kinetic energy is expressed by $1/2mv^2$, in which m = mass in grams and v = velocity in cm/sec. The basic unit of kinetic energy is the erg, which is equivalent to gm^2/sec^2.

406. Crush and Translational Injuries

The drag forces of the blast winds are strong enough to displace even large objects such as vehicles or to cause collapse of large structures such as buildings. These can result in very serious crush injuries. Humans themselves can become a missile and be displaced a variable distance and at variable velocities depending upon the intensity of the drag forces and the nature of the environment. The resulting injuries sustained are termed translational injuries. The probability and the severity of injury are functions of the velocity of the human body at the time of impact. If a representative displacement distance of 3.0 meters is assumed, the impact velocities which would be associated with various degrees of injury can be calculated. These are shown in Table 4-IV. The table shows terminal or impact velocities associated with significant but nonlethal blunt injury. It also shows those velocities which are associated with a probability of lethality. The velocities in Table 4-IV can be equated against yield, and the ranges at which such velocities would be found can be calculated. These are given in Table 4-V.

Table 4-IV. Translational Injuries

Velocity*(m/sec)	Probability of blunt injuries & fractures	Probability of fatal injuries
2.6	>1%	-
6.6	~50%	>1%
17.0	99%	~50%
44.5	-	99%

*Velocities are based on solid impact with a nonyielding surface.

Table 4-V. Ranges for Selected Impact Velocities of a 70-kg Human Body Displaced by Blast Wind Drag Forces for Different Yield Weapons

Weapon yield (Kt)	Velocities*(m/sec)		
	2.6	6.6	17.0
1	0.38 km	0.27 km	0.19 km
10	1.0 km	0.75 km	0.53 km
20	1.3 km	0.99 km	0.71 km
50	1.9 km	1.4 km	1.0 km
100	2.5 km	1.9 km	1.4 km
200	3.2 km	2.5 km	1.9 km
500	4.6 km	3.6 km	2.7 km
1000	5.9 km	4.8 km	3.6 km

* These velocities are selected from those listed in Table 4 -IV. Data account for ground friction and consider only prone personnel.

SECTION III - THERMAL INJURY

407. Mechanism of Injury.

The thermal radiation emitted by a nuclear detonation causes burns in two ways, by direct absorption of the thermal energy through exposed surfaces (flash burns) or by the indirect action of fires caused in the environment (flame burns). The relative importance of these two processes will depend upon the nature of the environment. If a nuclear weapon detonation occurs in easily flammable surroundings, indirect flame burns could possibly outnumber all other types of injury.

408. Thermal Effects.

a. Thermal radiation travels in a straight line from the fireball, and the amount of energy which is available to act upon a given target area decreases rapidly with distance. The thermal flux in watts per square centimeter decreases approximately

with the square of the distance from the point of detonation. This attenuation with distance varies somewhat with the nature of the environment and the weather, since thermal radiation is easily reflected. However, the attenuating effect of even a heavy cloud cover is surprisingly small. Since thermal radiation travels in straight lines, objects between the fireball and any targets will tend to shield and protect them.

b. Close to the fireball the thermal output will be so great that all objects will be incinerated. Immediate lethality obviously would be 100% within this range and to some extent beyond. The actual range out to which overall lethality would be 100% will vary with yield, position of burst, weather, the environment and how soon those burned can receive medical care. The mortality rate among the severely burned is much greater without early resuscitative treatment.

409. Thermal Energy and Burns to Exposed Skin.

Two factors determine the degree of burn injury in a given situation. The amount of thermal energy per square centimeter and the duration of the thermal pulse. The dose of thermal radiation to exposed skin required to cause a flash second-degree bum will vary from less than 16.7 joules/cm 2 to more than 29.3 joules/cm 2 depending on the yield of the weapon (Table 4-VI). A larger dose is required with larger yield weapons because of the nature of the pulse. Megaton weapons have much longer thermal pulses with much more gradual rates of increase. There is time for the skin to dissipate some of the thermal energy; and therefore, more is required to produce a given degree of injury. However, it must be realized that the same degree of injury from a megaton weapon is seen at a much greater range and over a much greater area than would be the case with kiloton weapons. The difference in dose required to produce a given burn injury is not a significant factor when compared with the increase in overall probability of injury associated with increasing yield.

Table 4-VI. Factors for Determining Probability of Second-Degree Burns to Bare Skin

Yield of weapon	1 Kt	10 Kt	100Kt	1Mt	10 Mt
Range in kilometers for production of second-degree burns on exposed surfaces (air burst)*	0.78	2.1	4.8	9.1	14.5
Duration of thermal pulse (sec)**	0.2	0.6	1.6	4.4	12.0
Joules/cm^2 required to produce second-degree burns on exposed skin	16.7	18.8	22.1	26.3	29.3

* Ranges calculated considering a 10-km visibility.
** Time for delivery of 70% of thermal energy.

410. Flash Burns Under Clothing.

While most thermal injury predictions are referred to exposed skin, it is important to remember the protection from burn that can be achieved with clothing. That protection, however, is not absolute. At temperatures below those required to ignite clothing, it is possible to transfer sufficient thermal energy across clothing to the skin to produce flash burns. The amount of heat energy conducted across clothing is a function of the energy absorbed by and the thermal conducting properties of the clothing. It will also be a function of whether the clothing is tight fitting or loose. Two uniform combinations have been specifically tested to determine the incident thermal exposure necessary to produce second-degree burns to skin under clothing. Table 4-VII summarizes the thermal burn criteria for skin under the U. S. Army summer uniform and the U. S. Army chemical protective overgarment. As can be seen by comparison with Table 4-VI, clothing significantly reduces the effective range to produce second-degree burns, thus affording significant protection against thermal flash burns. It should be noted that, because of the modifying effect of the uniforms, the exposures necessary to cause second-degree burns beneath the uniforms are yield independent.

*Table 4-VII. Incident Exposure Necessary to Cause Second-Degree Burns Under Clothing**

Clothing**	All yields - joules/cm^2
U.S. Army summer uniform (fatigue uniform and undershirt)	62.7
U.S. Army chemical protective overgarment (battledress overgarment, fatigue uniform, and undershirt)	129.7

* This is based on a 3mm separation between clothing and skin.
** The U.S. fatigue uniform is made from 50% cotton and 50% nylon. The battledress overgarment shell is 50% nylon and 50% cotton, and the lining is 100% synthetic material impregnated with a charcoal slurry. Fluence required to produce second-degree burns under uniforms remains constant as yield varies.

411. Flame Burns.

Indirect or flame burns result from exposure to fires caused by the thermal effects in the environment, particularly from ignition of clothing. This could be the predominant cause of burns depending on the number of and characteristics of (e.g., man-made fibers) flammable objects in an environment. This is particularly true for the large yield weapons, which can cause conflagrations and fire storms over extensive areas. Complications arise in the treatment of skin burns which have been created, in part, by melting man-made fibers; therefore, it may be advisable for clothing made of natural fibers to be worn next to the skin. The probability of flame burns cannot be quantified

with range as well as can flash burns. The variables of environmental flammability are too great to allow prediction of either incidence or severity. The burns themselves will be far less uniform in degree and will not be limited to exposed surfaces. For example, the respiratory system may be exposed to the effects of hot gases produced whenever extensive fires occur. Respiratory system burns are associated with severe morbidity and high mortality rates. Depending on the flammability of the material, blast winds may extinguish or fan the burning material.

412. Eye Injuries.

The effects of thermal/visual radiation on the eyes fall into two main categories, temporary flash blindness and permanent retinal scarring.

a. *Flash Blindness.*

(1) Flash blindness is caused by the initial brilliant flash of light produced by the nuclear detonation. More light energy is received on the retina than can be tolerated, but less than is required for irreversible injury. The retina is particularity susceptible to visible and short wavelength infrared light, since this part of the electromagnetic spectrum is focused by the lens with concentration of energy at the retinal surface. The result is bleaching of the visual pigments and temporary blindness.

(2) During the daylight hours, flash blindness does not persist for more than about 2 minutes, but generally is of the order of seconds. At night, when the pupil is dilated for dark adaptation, flash blindness will affect personnel at greater ranges and will last for longer periods of time (Figure 4-I). Partial recovery, such that personnel could function in lighted areas, may be expected within 3 to 10 minutes. Impairment of dark adaptation and night vision will persist for longer periods, however, and may seriously reduce combat effectiveness. It may require 15-35 minutes for recovery of night adaptation, depending upon the amount of light energy absorbed.

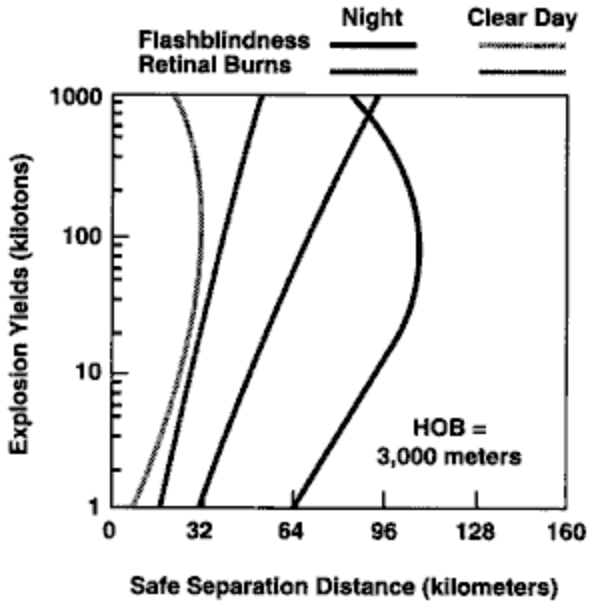


Figure 4-I. Flashblindness and Retinal Burn Safe Separation

(3) Figure 4-I illustrates flashblindness and retinal burn safe separation distances for an observer on the ground, as a function of explosion yield, for burst heights of 3,000 meters at night and on a clear day. Safe separation distances are those distances beyond which persons on the ground would not receive incapacitating eye injuries.

b. *Retinal Scarring*. A retinal burn resulting in permanent damage from scarring is also caused by the concentration of direct thermal energy on the retina by the lens. It will occur only when the fireball is actually in the individual's field of vision and would be a relatively uncommon injury. Retinal burns, however, may be sustained at considerable distances from the explosion (Figure 4-I). The apparent size of the fireball, a function of yield and range will determine the degree and extent of retinal scarring. The location of the scar will determine the degree of interference with vision, with a scar in the central visual field being potentially much more debilitating. Generally, a limited visual field defect, which will be barely noticeable, is all that is likely to occur.

CHAPTER 5

BIOPHYSICAL AND BIOLOGICAL EFFECTS OF IONIZING RADIATION

SECTION I - GENERAL

501. Introduction.

a. This chapter will cover basic biophysical and biological effects of ionizing radiation in order to form a foundation for understanding the clinical aspects of radiation injury discussed in Section IV of Chapter 6. This extended discussion of radiation does not imply that nuclear radiation will be the most important cause of casualties after a nuclear explosion. Blast and thermal injuries in many cases will far outnumber radiation injuries. However, radiation effects are considerably more complex and varied than are blast or thermal effects and are subject to considerable misunderstanding. As a result, a more detailed discussion is warranted. Since data from human experience are limited, much of the information in this chapter is based upon experimental information from animal studies.

b. A wide range of biological changes may follow the irradiation of an animal, ranging from rapid death following high doses of penetrating whole-body radiation to an essentially normal life for a variable period of time until the development of delayed radiation effects, in a portion of the exposed population, following low dose exposures. The nature and severity of these changes will depend upon a great variety of biological and physical factors. There are significant variations in response to irradiation associated with differences in species, age, and other biological factors, as well as the physical factors of dose, dose rate, or nature of the radiation. However, the biological responses to radiation are not unique. They fall within the range of standard tissue responses seen following other types of injury and occur as a result of similar biochemical and/or cell kinetic disturbances. As a result, the wide range of effects which is possible can be organized into a predictable scheme, the details of which form the basic material of this chapter.

SECTION II - BASIC BIOPHYSICAL ACTION OF IONIZING RADIATION

502. Nuclear Radiation.

A wide variety of ionizing radiation can interact with biological systems, but there are only four types of radiation associated with atmospheric and underground nuclear detonations of biological significance. In order of importance, they are gamma,

neutron, beta, and alpha. The physical natures of these are discussed at length in Chapter 2. However, certain aspects of their mechanisms of interaction with living tissue are summarized here.

503. Gamma Radiation.

a. Gamma radiation, emitted during the nuclear detonation or later in fallout, is highly energetic and is so penetrating that a significant part will pass through the human body without interaction. About 75% of the photons will interact with and lose energy to the atoms of the target tissue. This energy deposition may occur anywhere along a given photon's path, and therefore, anywhere in the body. If the gamma photon flux is high and the whole body is exposed, a fairly homogeneous deposition of energy will occur. This is in marked contrast to the highly localized energy deposition patterns of alpha and beta radiations.

b. Because of its penetrating ability, the effects of gamma irradiation can be independent of the location of the source, (i.e., internal or external to the body). High-energy gamma emitters deposited within the body can result in total body irradiation just as effectively as external sources, if the quantities deposited are large enough and despite the fact that the emitters may not be distributed uniformly throughout the body.

504. Neutron Radiation.

a. *Neutron Interaction.*

(1) Since neutrons are uncharged particles and can react only with the nuclei of target atoms, the probability of interaction of neutrons in the energy range characteristic of the fission spectrum detonation during their path through the human body is roughly comparable to that of low-energy gamma photons. Therefore, neutron radiation can result in whole-body irradiation. The energy deposition will not be uniform, and the side of the body which faces the detonation will absorb more energy than the opposite side. However, this difference, although of great theoretical interest, is not of operational importance. The major effect of this nonuniform deposition of energy will be to cause a wide variation in the typical radiation doses causing radiation sickness rather than significant variation in the overall clinical effects.

(2) As noted above, neutrons, since they are uncharged neutral particles, do not interact with the orbital electrons of atoms as do other forms of radiation. Instead, they interact with atomic nuclei directly. Because of their mass and energy, neutrons can cause severe disruptions in atomic structure, typically

causing a recoil "escape" of a target nucleus from its orbital electrons. This is much more common with the very light atoms, particularly hydrogen, since the mass of the photon making up the nucleus of common hydrogen is the major target atom in living tissue. When the nuclei of these latter are accelerated they are capable of causing dense ionization along their paths.

(3) In biological material, elastic collisions of this type between neutrons and the nuclei of light-weight atoms predominate. Due to their short range, the accelerated nuclei produced by these collisions will expend their energy along short tracks of high excitation and ionization density. In tissue, about 70% to 85% of the entire fast neutron energy is transferred to recoil hydrogen nuclei. The remainder of the neutron energy is dissipated in recoil nuclei of the other atoms noted above.

(4) After the neutrons have lost most of their energy through these collisions, they will reach an equilibrium energy state in which they are referred to as thermal neutrons. Such relatively slow moving neutrons have a high probability of being captured by the nuclei of a wide variety of elements such as sodium. The resulting materials are radioactive and generally decay rapidly. The resulting tissue irradiation is not a significant factor in radiation injury since the total energy released by the decay of these radioactive materials is extremely small compared to the total energy absorbed from the neutrons by elastic collisions. However, the quantities can be measured and can be used to estimate neutron doses in limited numbers of casualties.

b. *Neutron Relative Biological Effectiveness.*

(1) Relative biological effectiveness represents the effectiveness of a given radiation, compared to a reference radiation, (250 kilovolts (Kvp) x-rays), in producing the same level of response. Relative Biological Effectiveness (RBE) is defined as the ratio of the absorbed dose of a reference radiation to the absorbed dose of a test radiation to produce the same level of biological effect, other conditions being equal. (See Table 5-1.) When two radiations produce a biological effect that is not of the same extent and/or nature, the RBE cannot be specified.

Table 5-I. Relative Biological Effectiveness

Radiation	RBE
X-rays	1
Gamma rays	1
Beta particles	1
Alpha particles (into the body)	10 to 20
Neutrons:	
For immediate radiation injury	1
For cataracts, leukemia and genetic changes	4 to 10

(2) Marked changes in behavior, vomiting, cardiovascular disorders, neurological symptoms, and other symptoms have been observed in monkeys irradiated at doses between 0.5 and 6.5 gray (Gy) by a fission neutron flux with neutron dose/gamma dose ratios varying from 1 to 12 and a dose rate close to those delivered by "conventional" nuclear weapons.

(3) It was found that the neutron RBE (fission spectrum neutrons) for these disturbances was approximately between 0.5 and 1.2 in the range from 0.5 to 6.5 Gy. These RBE values must be confirmed by using a gamma radiation source with a dose rate comparable to that delivered by the reactors used and compared with those which would be obtained with neutrons from a fusion weapon. The above results lay particular stress upon the importance of intermediate dose and the biological effects of these as causes of incapacity can no longer be regarded as insignificant. In operational terms, neutron RBE varies with neutron energy, with neutron dose (the size of the neutron dose/gamma dose ratio), the dose rate and above all the dose gradient, particularly for determination of hematological LD50, but doubtless also for vomiting and early transient incapacitation (ETI). The RBE for ETI has been established as being equal to 1, because insufficient evidence has been collected to indicate otherwise. Relating dose to radiation effects in humans and other large mammals is further complicated by the fact that mixed-spectrum radiations change as they interact with body tissue. This change in quality of a mixed-spectrum field is significant since the biological damages produced by high-LET and low-LET radiations are not equivalent. High-LET radiations

such as alpha particles or fast neutrons are generally regarded to have a greater relative biological effectiveness than low-LET radiations such as x-rays and gamma photons. The one exception to this generalization that seems to be significant in predicting the effects of ionizing radiation on combat personnel is that gamma photons have been found to be more effective in producing early transient incapacitation than either high-energy neutrons or fission spectrum neutrons.

505. Beta Radiation.

a. High speed electrons in the form of beta radiation lose most of their energy after penetrating only a few millimeters of tissue. If the beta emitting material is on the surface of the skin, the resulting beta irradiation causes damage to the basal stratum of the skin. The lesion is similar to a superficial thermal burn. However, if the beta material is incorporated internally, the beta radiation can cause much more significant damage. The damage will be in spheres of tissue around each fragment or source of radioactive material. The total damage is a function of the number of sources and their distribution in the body. The distribution is determined by the chemical nature of the material.

b. Table 5-II lists the critical ranges of radiation exposure in tissue for beta emitters of various energies. These ranges are considerably greater than those for alpha particles (Table 5-III). In addition to a difference in range when compared with alpha radiation, there is also a significant difference in the pattern of energy deposition. The density of energy deposited is much less for beta irradiation than for alpha, and as a result, the target cells may be damaged rather than killed outright. Damaged cells may be of greater significance to the total organism than killed cells, particularly if they go on to become malignant or otherwise malfunction. Killed cells are replaced quickly in most tissues with any degree of reserve capacity and do not cause significant overall clinical effects unless the cells involved are highly critical or the fraction of cells killed in a given organ is large.

Table 5-II. Tissue Dose Rate at Various Distances Around a 37 KBq (1μCi) Particle of Various Beta Emitting Materials (Range in Tissue 1-10 mm)

Distance	Dose rate		
	^{14}C	^{90}SR - ^{90}Y	^{32}P
10μm	2,000,000	766,400	380,000
100μm -0.1 mm	1,500	7,380	3,700
200μm - 0.2 mm	40	1,705	930
400μm - 0.4 mm	0.03	340	230
600μm -0.6 mm	0	130	100
1,000μm -1.0 mm	0	34	30
10,000μm -10.0 mm	0	0.02	0
Max. beta energy (MeV)	0.156	0.546-2.27	1.71

Table 5-III. Tissue Dose Rate at Various Distances from a 37 KBq (1μCi) Alpha Emitter

Distance (μm)	Dose rate at distance (cGy/hr)
10	1.7×10^8
20	5.2×10^7
30	0

506. Alpha Radiation

a. The energy of these relatively heavy, positively charged particles is fully absorbed within the first 20 micrometers of an exposed tissue mass. If the source of the radiation is external, all of the alpha radiation is absorbed in the superficial layers of dead cells within the stratum corneum. If anything, even tissue paper, is interposed, the alpha particles will be absorbed, and not reach the skin. Because of this, alpha radiation is not an external hazard. If alpha emitting material is internally deposited, all the radiation energy will be absorbed in a very small volume of tissue immediately surrounding each particle. Alpha radiation has such limited penetrating ability that the maximum range for the highest energy alpha particle in tissue is less than 100 micrometers. Thus, while extremely high radiation doses may be deposited in the few cells immediately surrounding a source of alpha radiation, regions outside this small irradiated spherical volume are not affected. Table 5-III illustrates this for a 37 KBq (1.0 ❑Ci) source of an alpha emitter of moderate energy.

b. Beyond a radius of about 20 micrometers, the deposition of energy is very small. Due to the high radiation doses within this critical radius, the cells immediately

adjacent to the source are killed. They would then be removed by phagocytosis or replaced by fibrosis. Relatively little damage to the intact organism results, unless these cells are themselves highly critical. Most tissues with a reasonable reserve can tolerate the loss of a few cells quite readily, particularly if the tissues have a normally high turnover rate. Therefore, although internal alpha radiation can be lethal to individual cells, the overall acute hazard is small. Internal deposition of alpha particles are of importance on a long term basis in terms of causing radiation injury which is of greater significance than from beta particles. However, injury from internal deposition of alpha particles is not of military importance.

c. However, many alpha emitting materials also emit gamma radiation, and this gamma radiation may cause significant tissue injury, even though the total alpha energy exceeds the total gamma energy and the ratio of gamma emissions per alpha is very small. This follows from the fact that the penetrating power of gamma radiation is many times greater than that for alpha radiation so that the total volume of tissue exposed to damaging radiation is many times greater.

507. Radiochemical Action.

a. When radiation interacts with target atoms, energy is deposited, resulting in ionization or electron excitation as described in Chapter 2. This ionization or excitation must involve certain critical molecules or structures in a cell in order that the damage caused by radiation may follow the consistent patterns it does. It has been theorized that this localization of absorbed energy in critical molecules could be either a direct or an indirect action, i.e., the energy deposited by the radiation may involve particular sensitive chemical bonds directly, or it may be deposited elsewhere first and transferred to the sensitive bonds by means of an appropriate energy transfer system. The former mechanism implies that the radiation quite precisely hits particular target atoms, whereas the latter implies that there is a method for preferentially directing randomly deposited energy to sensitive sites.

b. The exact radiochemical mechanism involved in mammalian systems subjected to whole-body doses of penetrating radiation is not fully understood. However, the most reasonable hypothesis at the present time is that water, both intracellular and extracellular, is the primary site of radiation energy deposition and that the energy deposited in the water would be transferred to and affect sensitive molecules indirectly.

SECTION III - CELLULAR EFFECTS OF IONIZING RADIATION

508. General.

Observed cellular effects of radiation, whether due to direct or indirect damage, are basically similar for different kinds and doses of ionizing radiation.

a. *Cell Death*. One of the simplest effects to observe is cell death, the course of which can be described by various terms.

(1) *Pyknosis*. The nucleus becomes contracted, spheroidal, and filled with condensed chromatin.

(2) *Karyolysis*. The nucleus swells and loses its chromatin.

(3) *Protoplasmic Coagulation*. Irreversible gelatin formation occurs in both the cytoplasm and nucleus.

(4) *Karyorrhexis*. The nucleus becomes fragmented and scattered throughout the cell.

(5) *Cytolysis*. Cells swell until they burst and then slowly disappear.

b. *Changes in Cell Function*. Nonlethal changes in cellular function can occur as a result of lower radiation doses. These include delays in certain phases of the mitotic cycle, disrupted cell growth, permeability changes, and changes in motility.

(1) *Mitotic Cycle*. Mitosis may be delayed or inhibited following radiation exposure. Dose dependent inhibition of mitosis is particularly common in actively proliferating cell systems. This inhibition occurs approximately 40 minutes before prophase in the mitotic cycle, at a time when the chromosomes are discrete, but prior to the breakdown of the nuclear membrane. Subsequent irradiation after this radiation transition point does not delay mitosis. Delays in mitosis can cause profound alterations in cell kinetic patterns resulting in depletions of all populations. This is the basic kinetic patterns resulting in depletions of all populations. This is the basic mechanism underlying the later clinical changes seen in the hematopoietic and gastrointestinal syndromes of whole-body irradiation.

(2) *Disruptions in Cell Growth*. Cell growth may also be retarded, usually after a latent period. This may be due to progressive formation of inhibitory metabolic products and/or alterations in the cell microenvironment.

(3) *Permeability Changes*. Irradiated cells may show both increased and decreased permeability. Radiation changes within the lipid bilayers of the membrane may alter ionic pumps. This may be due to changes in the viscosity

of intracellular fluids associated with disruptions in the ratio of bound to unbound water. Such changes would result in an impairment of the ability of the cell to maintain metabolic equilibrium and could be very damaging even if the shift in equilibrium were quite small.

(4) *Changes in Cell Motility*. The motility of a cell may be decreased following irradiation. However, the presence of normal motility does not imply the absence of radiation injury. Irradiated spermatozoa, for example, may retain their motility and be capable of fertilization while carrying radiation-induced genetic changes which may alter subsequent embryogenesis.

509. Relative Cellular Radiosensitivity

In general, actively proliferating cells are most sensitive to radiation. On the other hand, the mitotic activity of all cells decreases with maturation. Thus, cellular radiosensitivity tends to vary inversely with the degree of differentiation. Cells may be classified functionally and in decreasing order of sensitivity into four categories: vegetative cells, differentiating cells, totally differentiated cells, and fixed non-replicating cells.

a. *Vegetative Cells*. These cells, comprising differentiated functional cells of a large variety of tissues, are generally the most radiosensitive. Examples include:

(1) Free stem cells of hematopoietic tissue (hemocytoblasts, primitive lymphoblasts, primitive erythroblasts, and primitive myeloblasts).

(2) Dividing cells deep in the intestinal crypts.

(3) Primitive spermatogonia in the epitheliums of the seminiferous tubules.

(4) Granulosa cells of developing and mature ovarian follicles.

(5) Basal germinal cells of the epidermis.

(6) Germinal cells of the gastric glands.

(7) Large and medium sized lymphocytes.

(8) Small lymphocytes, which are not included normally in this class of cells, but which are also highly radiosensitive.

(9) Mesenchymal cells.

b. *Differentiating Cells*. These cells are somewhat less sensitive to radiation. They are relatively short-lived and include the first generation produced by division of the vegetative mitotic cells. They usually continue to divide a limited number of times and differentiate to some degree between divisions. As differentiation occurs, radiosensitivity decreases. The best examples of this type of cell are the dividing and differentiating cells of the granulocytic and erythrocytic series in the bone marrow. This type also includes the more differentiated spermatogonia and spermatocytes in the seminiferous tubules and the ovocytes.

c. *Totally Differentiated Cells*. These cells are relatively radioresistant. They normally have relatively long lifespans and do not undergo regular or periodic division in the adult stage, except under abnormal conditions such as following damage to or destruction of a large number of their own kind. This class includes hepatocytes, cells of interstitial gland tissue of the gonads, smooth muscle cells, and vascular endothelial cells.

d. *Fixed Nonreplicating Cells*. These cells are most radioresistant. They do not normally divide, and some types, such as neurons, do not divide under any circumstances. They are highly differentiated morphologically and highly specialized in function. Cells in this group have widely varied life-spans and show progressive aging. This group includes the long-lived neurons, striated muscle cells, short-lived polymorphonuclear granulocytes and erythrocytes, spermatids and spermatozoa, and the supeficial epithelial cells of the alimentary tract.

510. Relative Organ Radiosensitivity.

The relative sensitivity of an organ to direct radiation injury depends upon its component tissue sensitivities. Table 5-IV lists various organs in decreasing order of radiosensitivity on the basis of a relatively direct radiation effect, parenchymal hypoplasia.

Table 5-IV. Relative Radiosensitivity of Various Organs Based on Parenchymal Hypoplasia

Organs	Relative radiosensitivity	Chief mechanism of parenchymal hypoplasia
Lymphoid organs; bone marrow; testes & ovaries; small intestines	High*	Destruction of parenchymal cells, especially the vegetative or differentiating cells
Skin; cornea & lens of eyes; gastrointestinal organs: cavity, esophagus, stomach, rectum	Fairly high	Destruction of vegetable and differentiating cells of the stratified epithelium
Growing cartilage; the vasculature; growing bones	Medium	Destruction of proliferating chondroblasts or osteoblasts; damage to the endothelium; destruction of connective tissue cells & chondroblasts or osteoblasts
Mature cartilage or bone; lungs; kidneys; liver; pancreas; adrenal gland; pituitary gland	Fairly low	Hypoplasia secondary damage to the fine vasculature and connective tissue elements
Muscle; brain; spinal cord	Low	Hypoplasia secondary damage to the fine vasculature and connective tissue elements, with little contribution by the direct effects on parenchymal tissues

*Embryonic tissue is also highly radiosensitive.

511. Radiation-Induced Chromosome Damage.

a. Cell nuclei contain chromosomes which in turn contain the genes controlling cellular somatic and reproductive activity. These chromosomes are composed of deoxyribonucleic acid (DNA), the macromolecule containing the genetic information. This is a large, tightly coiled, double-stranded molecule and is sensitive to radiation damage. Radiation effects range from complete breaks of the nucleotide chains of DNA, to point mutations which are essentially radiationinduced chemical changes in the nucleotides which may not affect the integrity of the basic structure. Intermediate effects, such as abnormal bonding between adjacent molecules and alterations in viscosity, have also been observed.

b. After irradiation, chromosomes may appear to be "sticky" with formation of temporary or permanent interchromosomal bridges preventing normal chromosome separation during mitosis and transcription of genetic information. Unequal division

of nuclear chromosome material between daughter cells with production of nonviable abnormal nuclei may result.

512. Genetic Effects.

Laboratory studies in animals indicate increased mutation rates with small doses of radiation. As radiation dose increases, mutation induction also increases. Mutations per unit dose decrease at low dose rates. However, viable mutations are still extremely rare. Most of the mutations are lethal and thus self-limiting. It must be kept in mind that radiation doses increase natural mutation rates and that the mutations produced, and not visibly detected, are permanent in regard to future generations.

513. Cell Kinetic Effects.

a. Each of the numerous cell renewal systems making up an animal's total cellular mass is normally in an equilibrium state between cell formation, proliferation, maturation, and death. Some systems, such as the adult central nervous system in higher animals, are stabilized at the end point of maturation, and the functional cells of such a system are not replaced if lost or destroyed. Other organ systems, such as the liver, which do not normally replace cells at a rapid rate, have the potential to regenerate large numbers of cells if needed. Other organ systems, such as the skin, the reproductive system, the gastrointestinal tract, and the hematopoietic system in the bone marrow, maintain a continuous high cell turnover rate. Bone marrow also has a large reserve capacity in the adult. A large fraction of it is normally nonfunctioning but has the potential to be functional if required. Failure of a particular organ system may or may not lead to death of the animal, depending on the importance of that system's functions, i.e., failure of gonadal function would not be lethal, whereas failure of bone-marrow function would be.

b. Regardless of the biophysical processes involved, one of the major biological effects of whole-body radiation, in the dose ranges causing the syndromes of bone-marrow depression and gastrointestinal damage, is a profound disturbance in the cell kinetics of these systems. Both the hematopoietic and the gastrointestinal system have fairly rapid cellular replacement rates and normally contain cell populations in all stages of maturation and differentiation from primitive stem cells to mature functional cells.

c. The stem cells of the various cell lines of these systems are almost all relatively sensitive to radiation whereas the mature functional cells are relatively resistant. As a result, following radiation, injured stem cells are not likely to mature. When the mature cells die or are otherwise lost they will not be replaced and the overall population of cells in the system will be decreased. If the radiation injury is repairable,

recovery of the ability of a stem cell population to mature will result in a gradual return of a mature, functional population. If the damage is irreversibly severe, there will be no recovery.

514. Bone-Marrow Kinetics.

The bone marrow contains three cell renewal systems: the erythropoietic (red cell), the myelopoietic (white cell), and the thrombopoietic (platelet). The time cycles and cellular distribution patterns and postirradiation responses of these three systems are quite different.

a. Studies suggest that a phuripotential stem cell gives rise to these three main cell lines in the bone marrow. Beyond this stem cell, each cell renewal system consists of a stem cell compartment for the production of erythrocytes, leukocytes (lymphocytes, granulocytes, monocytes, etc.), or platelets, a dividing and differentiating compartment, a maturing (nondividing) compartment, and a compartment containing mature functional cells.

b. Research studies suggest that each of these cell renewal systems operates under the influence of regulating factors, primarily at the stem cell level, through a negative feedback system initiated in large measure by the level of mature circulating cells in the peripheral blood. Normally, a steady-state condition exists between new cell production by the bone marrow and the numbers of functional cells. Morphological and functional studies have shown that each cell line, i. e., erythrocyte, leukocyte, and platelet, has its own unique renewal kinetics. The time-related responses evident in each of these cell renewal systems after irradiation are integrally related to the normal cytokinetics of each cell system.

515. Erythropoietic.

a. The function of this cell renewal system is to produce mature erythrocytes for the circulation. The transit time from the stem cell stage in the bone marrow to the mature red cell ranges from 4 to 7 days, after which the life-span of the red cell is approximately 120 days. The immature forms, i.e., erythroblast and proerythroblast, undergo mitosis as they progress through the dividing and differentiating compartment. Because of their rapid proliferating characteristics they are markedly sensitive to cell killing by ionizing radiation. Cell stages within the maturing (nondividing) and functional compartments, i.e., normoblast, reticulocyte, and red cell, are not significantly affected by midlethal to lethal range doses. The death of stem cells and of those within the next compartment is responsible for the depression of erythropoietic marrow and, if sufficiently severe, is responsible together with hemorrhage for subsequent radiation-induced anemia. Because of the relatively slow

turnover rate, e.g., approximately 1 percent loss of red cell mass per day, in comparison with leukocytes and platelets, evidence of anemia is manifested subsequent to the depression of the other cell lines, provided that significant hemorrhage has not occurred.

b. The erythropoietic system has a marked propensity for regeneration following irradiation from which survival is possible. After sublethal exposures, marrow erythropoiesis normally recovers slightly earlier than granulopoiesis and thrombopoiesis and occasionally overshoots the base-line level before levels at or near normal are reached. Reticulocytosis, occasionally evident in peripheral blood smears during the early intense regenerative phase occurring after maximum depression, often closely follows the temporal pattern of marrow erythropoietic recovery. Although anemia may be evident in the later stages of the bone-marrow syndrome, it should not be considered a survival-limiting sequela.

516. Myelopoietic.

a. The function of the myelopoietic marrow cell renewal system is mainly to produce mature granulocytes, i.e., neutrophils, eosinophils, and basophils, for the circulating blood. Of these, the neutrophils, because of their role in combatting infection, are the most important cell type in this cell line. The stem cells and those developing stages within the dividing and differentiating compartment are the most radiosensitive. These include the myeloblast, progranulocyte and myelocyte stages. As with the erythropoietic system, cell stages within the maturing (nondividing) compartment and the mature functional compartment, i.e., granulocytes, are not significantly affected by radiation doses within the midlethal range. Three to seven days are normally required for the mature circulating neutrophil granulocyte to form from its stem cell precursor stage in the bone marrow.

b. Mature functional granulocytes are available upon demand from venous, splenic and bone-marrow pools. Following an initial increase in circulating granulocytes (of unknown etiology), these pools are normally depleted before granulocytopenia is evident soon after radiation-induced bone-marrow injury. Because of the rapid turnover of the granulocyte cell renewal system due to the short life-span of its cells (approximately 8 days), evidence of radiation damage to marrow myelopoiesis occurs in the peripheral blood within 2 to 4 days after whole-body irradiation. The brief latent period between the time of irradiation and the beginning depletion of circulating granulocytes is related to the transit time of the nonradiosensitive cells within the nondividing, maturing marrow compartment, i.e., metamyelocyte and band forms, during their development into mature circulating granulocytes. Maturation depletion of these stages in the absence of feed-in of the earlier radiosensitive stages damaged by radiation accounts for the granulocytopenia.

c. Recovery of myelopoiesis lags slightly behind erythropoiesis and is accompanied by rapid increases in numbers of differentiating and dividing forms in the marrow. Prompt recovery is occasionally manifested and is indicated by increased numbers of band cells in the peripheral blood.

517. Thrombopoietic.

a. The thrombopoietic cell renewal system is responsible for the production of platelets (thrombocytes) for the peripheral circulating blood. Platelets along with granulocytes constitute two of the most important cell types in the circulation, the levels of which during the critical phase after midlethal doses will markedly influence the survival or nonsurvival of irradiated personnel. Platelets are produced by megakaryocytes in the bone marrow. Both platelets and mature megakaryocytes are relatively radioresistant; however, the stem cells and immature stages are very radiosensitive. During their developmental progression through the bone marrow, megakaryocytic precursor cells undergo nuclear division without cell division. The transit time through the megakaryocyte proliferating compartment in humans ranges from 4 to 10 days. Platelets have a life-span of 8 to 9 days.

b. Although platelet production by megakaryocytes may be reduced by a high dose of ionizing radiation, the primary effect is on stem cells and immature megakaryocyte stages in the bone marrow. As with the erythropoietic and myelopoietic systems, the time of beginning depression of circulating platelets is influenced by the normal turnover kinetics of cells within the maturing and functional compartments. Early platelet depression, reaching thrombocytopenic levels by 3 to 4 weeks after midlethal range doses, occurs from killing of stem cells and immature megakaryocyte stages and from maturation depletion of maturing and functional megakaryocytes.

c. Regeneration of thrombocytopoiesis after sublethal irradiation normally lags behind both erythropoiesis and myelopoiesis. Supranormal platelet numbers which overshoot the preirradiation level have occurred during the intense regenerative phase in human nuclear accident victims. The mechanism of the prompt rapid recovery of platelet numbers after acute sublethal irradiation may be explained by the response of the surviving and regenerating stem cell pool to a human feedback stimulus from the acute thrombocytopenic condition. Accelerated differentiation and maturation of immature megakaryocytes as well as marked increases in size of megakaryocytes contribute to the intense platelet production and eventual restoration of steady-state levels. Blood coagulation defects with concomitant hemorrhage constitute important clinical sequelae during the thrombocytopenic phase of bone-marrow and gastrointestinal syndromes.

518. Gastrointestinal Kinetics.

In view of the vulnerability of the small intestine to radiation damage and the important role it plays in the gastrointestinal syndrome, the cell renewal kinetics of the villi of this segment are important.

a. The renewal system is in the crypt and villus where epithelial cell formation, migration and loss occur. The four cell renewal compartments are: stem cell and proliferating cell compartment, maturation compartment, functional compartment, and the extrusion zone. Stem cells and proliferating cells move from crypts into a maturing only compartment at the neck of the crypts and base of the villi. Functionally mature epithelial cells than migrate up the villus wall and are extruded at the villus tip. The overall transit time from stem cell to extrusion on the villus for humans is estimated as being 7 to 8 days. Shorter times for epithelial cell renewal systems have been reported in experimental animals.

b. Because of the high turnover rate occurring within the stem cell and proliferating cell compartment of the crypt, marked damage occurs in this region by whole-body radiation doses above the midlethal range. Destruction as well as mitotic inhibition occurs within the highly radiosensitive crypt and proliferating cell compartments within hours after high doses. Maturing and functional epithelial cells continue to migrate up the villus wall and are extruded albeit the process is slowed. Shrinkage of villi and morphological changes in mucosal cells, i.e., columnar to cuboidal to squamoid, occur as new cell production is diminished within the crypts. Continued extrusion of epithelial cells in the absence of cell production can result in denudation of the intestinal mucosa. Concomitant injury to the microvasculature of the mucosa and submucosa in combination with epithelial cell denudation results in hemorrhage and marked fluid and electrolyte loss contributing to shock. These events normally occur within 1 to 2 weeks after irradiation. A second mechanism of injury has recently been detected at the lower range of the gastrointestinal syndrome, or before major denudation occurs at higher doses of radiation. This response is a functional increase in fluid and electrolyte secretion on the epithelial cells without visible cell damage. This second mechanism may have important implications for fluid replacement therapy. Other secondary complications which contribute significantly to the gastrointestinal syndrome will be described elsewhere.

SECTION IV - SYSTEMIC EFFECTS OF WHOLE-BODY IRRADIATION

519. General.

Whole-body irradiation is the most important type of radiation exposure since it is the most damaging and is discussed in the greatest detail in this section. However, partial body and specific organ irradiation can occur, particularly from internal deposition and retention of radioactive fission products found in fallout. Basic biophysical

principles of internal irradiation are also discussed in a later section of this chapter. Severe radiation sickness is seen following large dose of external whole-body irradiation. Variable lesser degrees of radiation sickness may occur following partial body irradiation. The mechanisms underlying the various syndromes of severe radiation sickness are emphasized in this section.

520. Median Lethal Dose (LD_{50}).

a. *Lethality*.

(1) When comparing the effects of various types or circumstances, that dose which is lethal to 50% of a given population is a very useful parameter. The term is usually defined for a specific time, being limited, generally, to studies of acute lethality. The common time periods used are 30 days or less for most small laboratory animals and to 60 days for large animals and humans. On occasion, when a specific type of death is being studied, the time period used will be shorter. The specified period of time is indicated by a second number in the subscript; $LD_{50/30}$ and $LD_{50/5}$ indicate 50% mortality within 30 days and 5 days, respectively. The LD_{50} is a median; the easiest method of approximating it is by plotting experimental data on an appropriate graph and then estimating it by inspection. It should be understood that the $LD_{50/60}$ assumes that the individuals did not receive other injuries or medical treatment.

(2) Figure 5-I is a graphic representation of a typical mortality response to radiation. The curve drawn through the data points is sigmoid, indicating that the mortality response to increasing dose approximates a normal distribution. A sigmoid curve is difficult to plot, particularly when the number of data points is limited, and a preferred method which allows the plotting of experimental data along a straight line is used in most mortality studies. Figure 5-II shows the same experimental data plotted on a specially constructed graph, termed a probit graph. This distortion is deliberate and is based upon the function of a normal distribution, so that data from a normal distribution can be plotted on such a graph, which seems to fit the data. The LD50 can then be estimated by inspection. This method is simple and used extensively. However, it should only be used when it has been demonstrated that the dose response phenomenon being studied does indeed follow or at least reasonably approximates a normal distribution. There is controversy about what the $LD_{50/60}$ for humans is. A full discussion of this issue is beyond the scope of this overview.

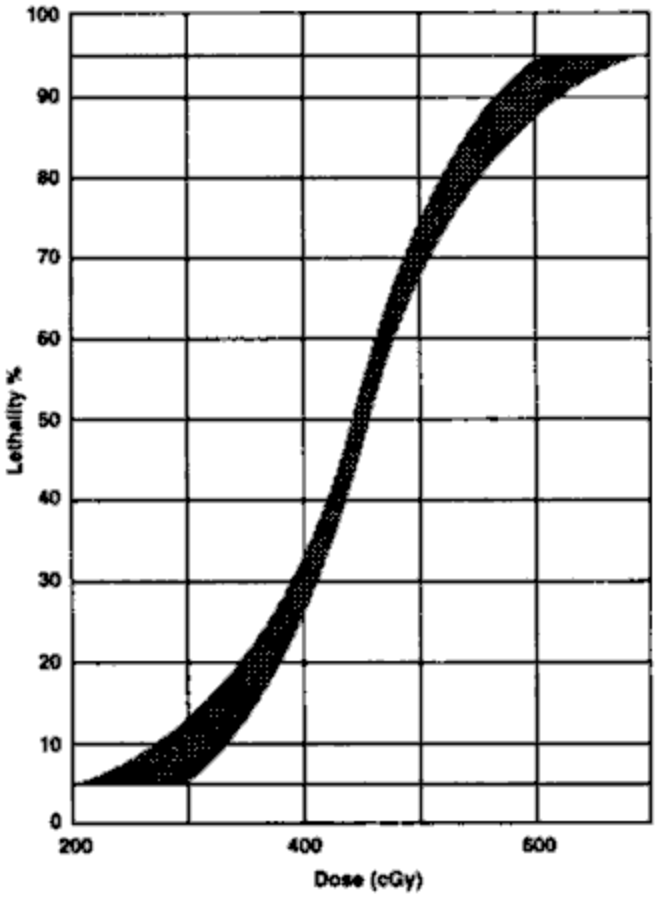


Figure 5-I. Typical Lethality as a Function of Dose

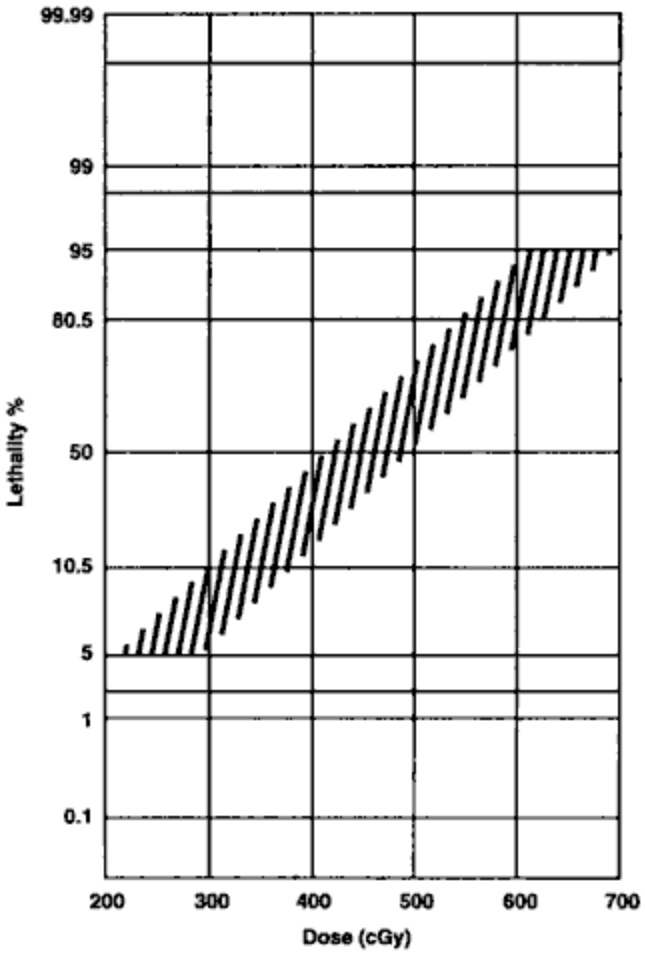


Figure 5-II. Lethality Response as a Function of Dose Obtained by Probit Analysis

(3) Medically, other figures of interest are the dose that will kill virtually no one, (LD5), and the dose that will kill virtually every one (LD95). Approximations of those doses are within the ranges 200-300 cGy (free in air) and 600-700 cGy (free in air), respectively.

b. *Radiation-Induced Early Incapacitation.*

(1) The focus of animal studies has been the incapacitation of the subhuman primate, since the incapacitation response has military relevance and the response of primates seems most like a human's response after acute wholebody irradiation. For high radiation doses (in excess of 1000 cGy), early transient incapacitation (ETI) occurs on average within 5 to 10 minutes after acute whole-body irradiation. With lowering the dose the median time of ETI occurrence increases up to 12 to 15 minutes. Typical duration of ETI is of the

order of 15 minutes. Performance decrement in the monkey has been evaluated for numerous behavioral takes after whole-body and partial-body irradiation for various radiation qualities and dose rates. Several generalizations have emerged from these studies.

(a) Early transient incapacitation is qualitatively very similar for many behavioral tasks.

(b) The frequency function of radiation of incapacitation within a population increases as a dose.

(c) Incapacitation can be elicited by either trunk-only or head-only irradiation.

(d) Neutrons are less effective in producing early transient incapacitation than are gamma photons. The relative biological effectiveness for incapacitation of neutrons to gammas has been estimated between 0.23 and 0.62.

(e) The frequency of incapacitation produced by a given radiation dose is proportional to the demands or stress of the task being performed. These findings and the data they represent are the basis for the current combat casualty criteria. The present criteria are based on the incapacitating dose levels for both physically demanding tasks and undemanding tasks. They do not include combat ineffectiveness due to partially degraded performance that may result from slower reaction to the task, task stress, or prodromal effects of the acute radiation sickness.

(2) For yields of 5-10 Kt (or less), initial nuclear radiation is the dominant casualty producer on the battlefield. Military personnel receiving an acute incapacitation dose (30 Gy) will become performance degraded almost immediately and combat ineffective within several hours. However, they will not die until 5-6 days after exposure if they do not receive any other injuries which make them more susceptible to the radiation dose. Soldiers receiving less than a total of 150 cGy will remain combat effective. Between those two extremes, military personnel receiving doses greater than 150 cGy will become degraded; some will eventually die. A dose of 530-830 cGy is considered lethal but not immediately incapacitating. Personnel exposed to this amount of radiation will become performance degraded within 2-3 hours, depending on how physically demanding the tasks they must perform are, and will remain in this degraded state at least 2 days. However, at that point they will experience a recovery period and be effective at performing nondemanding tasks for about 6

days, after which they will relapse into a degraded state of performance and remain so for about 4 weeks. At this time they will begin exhibiting radiation symptoms of sufficient severity to render them totally ineffective. Death follows at approximately 6 weeks after exposure. Experiments conducted with animal models have shown that exposure to high doses of ionizing radiation (of the order of 25 Gy) results in an immediate precipitous decline in cerebral blood flow (CBF) which is followed by a partial recovery at 20-30 minutes, and subsequent slower secondary decrease in CBF thereafter accompanied by parallel changes in systemic blood pressure. These data indicate that radiation adversely affects the ability of the brain to regulate its blood supply. The implication of this indication extends into the realm of behavioral studies of early transient incapacitation and performance decrement (ETI-PD). The activity of certain brain enzymes involved in neurotransmitter metabolism is also considerably affected during ETI.

(3) Experimental results from animal studies indicate that, in general, partial body shielding reduces the behavioral effects of radiation. Head shielding is more effective in preserving the behavioral performance after exposure than is trunk shielding. Head shielding not only reduces the incidence of incapacitation but reduces the incidence of convulsions that normally accompanies early incapacitation. In all experimental causes studied to date, head shielding is most effective for doses in excess of 25 Gy.

521. Reproductive Cell Kinetics and Sterility.

a. Despite the high degree of radiosensitivity of some stages of germ cell development, the testes and ovaries are only transiently affected by single sublethal doses of whole-body irradiation and generally go on to recover normal function. In male test animals, low doses of whole-body irradiation cause abrupt decreases in sperm counts. The degree of decrease is dose dependent, but a transient azoospermia can appear at sublethal radiation doses. The resulting sterility may last several months to several years, but recovery of natural fertility does occur. The recovery depends upon the regeneration of those elements of the stem cell population which were in a relatively resistant part of the germ cell cycle. Other data suggest that under some conditions new spermatogonia may be formed by transformation from more radioresistant fixed stem cells.

b. When chromosome aberrations are produced in somatic cells, the injury is restricted to the specific tissue or cell system. However, when aberrations occur in germ cells, the effects may be reflected in subsequent generations. Most frequently, the stem cells of the germ cell line do not develop into mature sperm cells or ova, and no abnormalities are transmitted. If the abnormalities are not severe enough to prevent

fertilization, the developing embryos will not be viable in most instances. Only when the chromosome damage is very slight and there is no actual loss of genetic material will the offspring be viable and abnormalities be transferable to succeeding generations. These point mutations become important at low radiation dose levels. In any population of cells, spontaneous point mutations occur naturally. Radiation increases the rate of these mutations and thus increases the abnormal genetic burden of future generations.

522. Recovery.

a. *Recovery Processes.*

(1) A variety of recovery processes may reduce radiation damage to a varying extent. For example, when a chromosome is broken, the broken ends tend to rejoin thus reconstituting the chromosome, but occasionally the broken ends seal over before rejoining thus leaving permanent chromosome damage. If two (or more) chromosomes are broken within the same cell, rejoining of inappropriate broken ends can occur and so may lead to permanent chromosomal change of a different kind. Repair of the broken ends of chromosomes, like all other repair processes following radiation damage, is not specific in respect of radiation damage. Repair is a biological process specific to a particular kind of damage which comes into play whatever the agent which causes that damage. These particular examples and others relating to DNA and its repair or to increased permeability of cell membranes, etc., are important in practice only for very large exposures.

(2) Three specific recovery processes are directly relevant to the medical aspects of defense operations. The first is an intracellular recovery within individual cells which have been sublethally irradiated. The second is a specific recovery of a specific tissue in which killed or damaged cells are replaced by division of surviving and minimally damaged or undamaged cells within that tissue, a process often called repopulation. Between them, these two processes may allow a complete return of function to normal. When the local dose is large enough, repair may be possible but incomplete. Repair of a specific tissue may be carried out without complete replacement of all the cells of the tissue. Healing may involve tissue atrophy and/or fibrosis and the irradiated tissue may be permanently scarred. The third, a combination of the first two types of recovery, can be very approximately quantified for lethality in humans by the use of the operational equivalent dose formula in cases where the irradiation period is protracted over several hours or longer as might happen in fallout conditions.

b. *Intracellular Recovery.* Individual irradiated cells have the ability to repair themselves as long as the amount of intracellular damage does not exceed a threshold value. The basic reason why sublethally irradiated cells survive and then recover is that a certain minimum amount of radiation energy must be deposited within a cell in order to kill it. Even when a mass of cells is uniformly exposed to low LET radiation, the amount of radiation energy deposited in individual cells is not the same for each cell but varies widely from cell to cell. As the dose increases, the proportion of cells increases in which a just lethal or more than lethal amount of energy is deposited. But all the other irradiated cells, those in which either no radiation energy or a sublethal amount is deposited, restore themselves to normal if given sufficient time to do so. Although controversial, it is generally believed that this mechanism for recovery is more effective in cells not undergoing active cell division, e.g., quiescent stem cells, than in cells undergoing active cell division, e.g., the basal cells of the intestinal crypts and the ordinary blast cells of the bone marrow. In quiescent cells, full recovery from sublethal radiation damage takes only a few hours. This can be demonstrated by dividing a dose into 2 fractions separated by a few hours when the damage observed will be less than when the whole dose is given all at once. This so-called Elkind repair continues during a protracted exposure to radiation, such as to fallout. It does not require a radiation-free interval.

c. *Repopulation.*

(1) Repopulation brought about by stem cell proliferation is a particularly important recovery mechanism in both the bone marrow and the gastrointestinal tract whenever the radiation exposure has been large enough to reduce cell numbers. Stem cells divide normally in both these tissues, because stem cell turnover is required to compensate for the normal continuously occurring removal of differentiated cells. Stem cell division can be accelerated by large doses of radiation. Large doses of radiation cause enough damage to stimulate this repopulation, just as any other severe insult would do. The effects of small doses are not recognized soon enough for accelerated proliferation to take place.

(2) In bone marrow, large microphage cells produce factors that either stimulate or shut down the stem cells that are the progenitors of the erythropoietic, granulopoietic, or thrombopoietic series of blood cells. The "factor producing" cells influence one another and depress the production of one factor while the opposite is being produced. Stem cell responses continue until the factor is changed. Some stem cells have the ability to cycle at faster rates than others but with lower efficiency, producing fewer mature cells eventually than the slower cycling cells. If the duration of exposure is sufficiently prolonged and the continued exposures are sufficiently large, then the repopulation process may

become less efficient. However, it may take several months before the repopulation process becomes significantly impaired and so it is not likely to be relevant in a short duration nuclear warfare scenario.

d. *Problems with Application of An Equivalent Dose Formula*. It has long been realized that it is desirable to quantify recovery from ionizing radiation damage, especially when received more or less continuously over a period of time as would be expected when operating in fallout conditions. For operational reasons, the quantification needs to be relatively simple to use and should not require a computation with parameters that could not be established in a nuclear warfare scenario. Consequently, several equivalent dose formulas have been proposed which estimate the lethal dose from accumulated exposure. As such, these formulas can be used as guides to predict the levels of external exposure that could be tolerated from fallout fields. On the battlefield, however, they are of very limited use and could lead to serious overestimates of combat capability because they do not account for the effects of neutron exposures and predict only lethality, not radiation sickness, which could severely impair the effectiveness of combat personnel. Current equivalent dose formulas are applicable to a very small portion of a battlefield population, because they are valid only for external gamma doses received at low dose rates. Therefore, they cannot be used to predict the response of anyone exposed to neutrons. This limitation renders the formulas unusable for any military personnel irradiated at the time of a nuclear detonation since neutron dose is known to be more lethal than a comparable dose of gamma radiation alone. Present formulas potentially would be applicable only to forces being introduced into a fallout field after the cessation of nuclear detonations. Their practical use on the battlefield is further reduced by NATO and enemy nuclear targeting doctrine which call for detonations at altitudes that preclude the generation of fallout and by the difficulty in predicting arrival of fallout fields. Given the small range of application to the nuclear battlefield and the possible errors they might cause, current equivalent dose formulas are inappropriate for operational decision making on the nuclear battlefield.

SECTION V - DELAYED EFFECTS

523. General.

Late or delayed effects of radiation occur following a wide range of doses and dose rates. Delayed effects may appear months to years after irradiation and include a wide variety of effects involving almost all tissues or organs. Some of the possible delayed consequences of radiation injury are life shortening, carcinogenesis, cataract formation, chronic radiodermatitis, decreased fertility, and genetic mutations.

524. Carcinogenesis.

a. Irradiation of almost any part of the body increases the probability of cancer. The type formed depends on such factors as area irradiated, radiation dose, age, and species. Irradiation may either increase the absolute incidence of cancer or accelerate the time or onset of cancer appearance, or both. There is a latent period between the exposure and the clinical appearance of the cancer. In the case of the various radiation-induced cancers seen in mankind, the latency period may be several years. Latency as well as the dose required to induce cancers varies with the cancer site and with the species studied. Latent periods for induction of skin cancers in people have ranged from 12 to 56 years after x irradiation therapeutic exposures with estimated doses of several thousand roentgens. Fifteen years is reported as a latent period for bone tumors from radium. This latency related to bone tumors is very dependent upon the dose and type of radiation emitted by the radionuclide.

b. A leukemogenic effect was expected and found among Hiroshima and Nagasaki survivors. Peak incidence occurred 6 years after exposure and was less marked for chronic granulocytic leukemia than acute leukemia. The incidence was inversely related to distance from the hypocenter. British persons receiving radiotherapy for spondylitis showed a dose response relationship for leukemia, with peak incidence occurring 5 years after the first exposure. Studies have demonstrated that ionizing radiation can induce more than one kind of leukemia in people, but not chronic lymphocytic leukemia.

c. Predisposing factors for tumor development include heredity, age, hormones, and prior exposure to physical trauma, chemical agents and ionizing radiation. The actual processes by which cancer is induced are not known. Somatic mutations, virus infections, and precancerous abnormalities in tissue organization and vascular supply have all been postulated.

525. Cataract Formation.

A late effect of eye irradiation is cataract formation. It may begin anywhere from 6 months to several years after exposure. While all types of ionizing radiation may induce cataract formation, neutron irradiation is especially effective in its formation, even at relatively low doses. Cataract formation begins at the posterior pole of the lens and continues until the entire lens has been affected. Growth of the opacity may stop at any point. The rate of growth and the degree of opacity are dependent upon the dose as well as the type of radiation. The threshold for detectable cataract formation in 2 Sv (sievert) (200 REM (roentgen equivalent, man)) for acute radiation doses and 15 Sv (1500 REM) for protracted doses.

526. Chronic Radiodermatitis.

Delayed, irreversible changes of the skin usually do not develop as a result of sublethal whole-body irradiation, but instead follow higher doses limited to the skin. These changes are a common complication in radiation therapy but they should be rare in nuclear combat unless there is heavy contamination of bare skin with beta emitter material from fallout, in which case beta-induced skin ulceration could be seen. The condition should be easily prevented with reasonable hygiene and would be particularly rare in climates where the soldiers were fully clothed (arms, legs, and neck covered). Table 5-V lists the degrees of radiation dermatitis for various radiation doses.

Table 5-V. Radiation Dermatitis

Radiation	Dose	Effect
Acute dose (mainly beta)	6 to 20 Sv (600 to 2000 REM)	Erythema only
	20 to 40 Sv (2000 to 4000 REM)	Skin breakdown in 2 weeks
	>300 Sv (30,000 REM)	Immediate skin blistering
Chronic doses	>20 Sv (2,000 REM)	Dermatitis, with cancer risk

SECTION VI - INTERNAL IRRADIATION

527. Introduction.

a. When radioactive materials are incorporated into the body and retained, significant radiation injury can be sustained by specific tissues in which the materials are concentrated or in some instances by the whole body. The primary factors which determine the type and degree of injury are the types and amounts of the isotopes deposited and the nature and energies of the radiation emitted.

b. Each isotope follows a fairly specific biological pathway in the body. This pathway may be quite complex with several compartments and is determined by the chemical nature of the isotope. A given isotope may be concentrated or retained in a specific organ or tissue during the time it is in the body. It may be eliminated from the body, and the rates of elimination of different isotopes vary considerably. More than one isotope may be incorporated in the body at the same time, and the effects of a mixture of isotopes found in fallout would be additive.

c. In this section, certain basic principles and factors governing isotopes in the body are discussed; these include their distribution, action, and elimination. The associated clinical problems are discussed in Chapter 6.

528. Incorporation of Radioactive Material.

The basic routes of entry for isotopes are: inhalation, ingestion, and absorption through the skin. Following ingestion or inhalation, a given material may be absorbed into the blood stream, depending upon its volubility. Insoluble materials are not absorbed, except in extremely small amounts, and may be eliminated fairly rapidly directly from the respiratory and gastrointestinal tracts. However, under certain circumstances, insoluble materials can be retained at or near the original site of deposition, e.g., in the lungs or in wounds, or may be translocated to regional lymph nodes, where again they will constitute an internal radiation hazard. Only the very small particles of radioactive materials, 10 microns in diameter or smaller, are deposited in the alveolar airsacs.

a. *Inhalation.*

(1) An insoluble material which is inhaled in the form of an aerosol will be deposited along the tracheobronchial tree. Much of it will be removed by the ciliary action of the mucosa lining most of the respiratory system, but a certain fraction, depending on the size, shape, and density of the particles, will penetrate down to the alveolar airsacs and remain. Only the very smallest particles penetrate that far; and so, the percentage of inhaled insoluble particles which are retained in the lungs is small, generally less than 25%. However, material so retained can be a considerable hazard to the lung, since it may remain for a long time. A portion of this material will be picked up by the lymphatic system draining the various pulmonary regions. It will then be collected by and remain in the lymph nodes of the lungs and still be a longterm hazard to lung tissue. A small fraction of the material may reach the blood stream and end up trapped in the reticuloendothelial system in various regions of the body and for certain isotopes, such as plutonium and strontium, also in bone.

(2) If a soluble material is inhaled, it is absorbed very rapidly and completely, and often will not remain in the lungs long enough to cause significant damage. Once in the circulation, it will be distributed in the body in the same way as it would following any other mode of entry.

b. *Ingestion.*

(1) An insoluble material which is ingested will remain in the gastrointestinal tract and become mixed in and part of the fecal material in the large bowel, with which it will then be eliminated. This includes swallowed material cleared from the upper respiratory tract and the tracheobronchial system by ciliary action. Insoluble material is not retained in the gut as it is in the lungs or in soft tissues, and the radiation hazard is limited in time to that required for transit and elimination, generally a matter of hours. As a result, the radiation hazard is negligible, unless the material includes a highly active gamma emitter. Normally, beta and alpha radiation from insoluble radioactive material in the gut lumen will not cause significant damage. The few cells of the mucosa which are damaged slough off and are replaced rapidly. A gamma emitter on the other hand would be a whole-body hazard as long as it was in the gut. Highly radioactive fallout containing fission products emitting beta and gamma radiations could cause some gastrointestinal tract damage if accidentally ingested with contaminated foodstuffs or water. However, in most such instances, the whole-body exposure received from external gamma radiation in the area would be the controlling hazard.

(2) When a soluble material is ingested, absorption is quite efficient. This is the most significant route of entry for the soluble isotopes in fallout, particularly when fallout-contaminated water or food is consumed. A number of fission products can become incorporated into vegetation and enter into complex food chains. In some instances, certain radioactive materials can be concentrated in these chains increasing the eventual hazard to humans.

c. *Transcutaneous Absorption.*

(1) An insoluble material contaminating the intact skin can be an external hazard only if it is a gamma or beta emitter. It will not be absorbed into the blood stream and thus will not become an internal hazard. Conceivably, contamination of the skin with large quantities of gamma emitting materials could result in significant whole-body irradiation. This could occur when personnel have been subjected to heavy fallout contamination. However, this can be easily prevented by prompt removal of contaminated clothing and washing exposed areas of skin. If a wound is contaminated, insoluble material will tend to remain localized in the tissue at the wound site, unless removed by debridement. Some would be present within the eschar. This type of contamination should not cause a serious problem, unless it is particularly high in radioactivity. A small but measurable fraction of the material will be cleared from the wound site by lymphatic drainage. Most of this material will be trapped in the regional lymph nodes which drain the area of the wound, similar to that process described for the lungs.

(2) Soluble material will be absorbed readily through wound sites and distributed within the body organs and tissues according to the usual metabolism of the stable isotope of the element in question. Some soluble materials, particularly tritium, will be absorbed rapidly and totally across the intact skin.

529. Elimination of Isotopes.

a. A radioactive material must be eliminated from the body to remove its hazard. Detoxification, which is effective against materials which are chemical hazards, will not be effective since radioactivity is not modified by chemical changes. The methods of elimination include renal excretion for most soluble materials, elimination in the feces for materials which are retained in the gut or which can be secreted in the bile, and exhalation for volatile materials and gases. Chelating agents, e.g., calcium or zinc DTPA (diethylenetriamine pentaacetic acid), if administered soon after exposure, are effective in enhancing the elimination of certain radioisotopes. These materials are not very effective for radioisotopes which have been incorporated and fixed in organs and tissues, e.g., bone. Under conditions of nuclear war, chelation therapy is very unlikely to be used. (See 717e.)

b. The rate at which a material is eliminated is usually expressed as the biological half-life. This is the time it takes for one-half of a given amount of material to be excreted or eliminated. During each successive half-life, an additional one-half is removed from the body. It is analogous, therefore, to the physical half-life. Not all materials follow a simple exponential elimination process, but this method of expression is sufficiently accurate to be applicable to most soluble isotopes. An exception which must be noted is the retention of insoluble heavy metals such as plutonium in the lungs and in bone. The rates of loss under these circumstances are not exponential and are very slow.

c. The biological half-time may be variable. A prime example of this is body water, the turnover of which can be as short as 4 days to as long as 18 days depending upon the state of hydration, volume of intake, and renal function. If tritiated water is incorporated into the body, the biological half-life is the factor determining the hazard since it is so much shorter than the physical half-life of about 12 years. Reduction of the biological half-life to a minimum by overhydration and the administration of diuretics has obvious value and is the recommended therapy in cases of exposures to tritium. Other isotopes cannot be cleared from the body as rapidly, and there is no adequate treatment available at present for increasing the rate of removal of a mixture of isotopes which would be incorporated into the body as a result of ingesting fallout contaminated food and water.

d. The overall hazard of materials which are eliminated exponentially will be a function of their physical and biological half-lives considered together. Whichever is shorter will become the primary factor. The effective half-life is usually determined and expressed by the following formula:

$$\text{Effective half-life} = \frac{\text{Biological half-life x Radiological half-life}}{\text{Biological half-life + Radiological half-life}}$$

e. The uptake by the body of radioisotopes can be blocked in some cases. For example, potassium iodide or iodate if given prior to or soon after an intake of radioiodine, will reduce the uptake of radioiodine by the thyroid gland. Similarly, orally administered Prussian Blue will reduce the absorption of cesium from the gut and Alginate will reduce strontium absorption. No policy exists which would allow for NATO forces to stock and issue chelators.

CHAPTER 6

GENERAL MEDICAL EFFECTS OF NUCLEAR WEAPONS: DIAGNOSIS, TREATMENT, AND PROGNOSIS

SECTION I - GENERAL

601. Introduction.

This chapter covers clinical aspects of the various medical problems which may be seen in modem warfare as a result of the use of nuclear weapons. Blast, thermal, and radiation injuries are discussed first. Combined injury is discussed as a separate subject because of the special problems which patients present when radiation sickness complicates other serious injuries. The psychological and public health aspects of nuclear warfare are also combined.

SECTION II - BLAST INJURIES

602. General.

The types of blast injuries by nuclear weapons are more varied than those caused by conventional weapons and are the result of two basic mechanisms, either the direct action of the blast wave overpressures or the indirect action of flying debris or violent displacement of individuals against other objects. In addition, the blast injuries caused by nuclear weapons will frequently be complicated by associated thermal and/or radiation injuries. Finally, the number of casualties produced at any one time in a given area will be very much greater for nuclear weapons than for conventional weapons.

603. Diagnosis.

The treatment of blast injuries is generally not difficult unless there is unrecognized internal injury with slow hemorrhage. As noted, missile injuries will predominate. About half of the patients seen will have wounds of their extremities. The thorax, abdomen, and head will be involved about equally. Missile injuries of the thorax, neck, and the head will be responsible for a large percentage of deaths because these types of injuries have a high probability of immediate fatality. The missile injuries caused by nuclear weapons will, in general, be of the low velocity type, and surprisingly severe injuries may be survived since extensive soft tissue cavitation

would not be a factor. These injuries can occur with or without perforating wounds of the abdomen or the chest.

604. Treatment.

The treatment of blast injuries, whether combined with other injuries or not, is best managed by applying accepted principles of combat surgery. Treatment is divided into four basic plans:

a. *Resuscitative Phase.* Lifesaving resuscitative measures designed to prepare the patient for definitive surgical treatment come first. These include the establishment of the airway assuring the adequacy of respiration, replacement of lost blood and fluids, and splinting of possible fractures, particularly those involving the cervical vertebrae. Some resuscitative measures must be started prior to evacuation from the battlefield, particularly if ground transportation is used rather than helicopter evacuation.

b. *Surgical Phase*. Definitive surgery should be done after resuscitative measures have been used to improve the patient's condition in order to minimize the risk of surgery and anesthesia. Occasionally, lifesaving surgery must be done without delay, but normally there is time to prepare patients for surgery if they have survived long enough to reach a treatment facility.

c. *Recovery Phase*. In the immediate postoperative period, patients require minimal movement. Transportation to other facilities should be delayed until the patient's condition has stabilized.

d. *Convalescent Phase.* Patients in this phase of treatment should be evacuated back to specialized convalescent facilities in order to keep the patient load of forward surgical hospitals as low as possible. Many injuries may require a prolonged recovery period before the individual has recovered to the point where he/ she can resume their duties. Both the convalescent and recovery phases will be more protracted with the addition of the radiation injury.

SECTION III - THERMAL INJURIES

605. General.

Many burn casualties may occur as a result of incendiary attacks on cities and military personnel in the field during conventional warfare. However, in nuclear warfare, burns could become the most frequent injury seen. Because of the complexity of burns treatment and the increased logistical requirements associated with the management of burns, they will constitute the most difficult problem faced by the medical service.

606. Diagnosis.

Certain factors are of prime importance in the early evaluation of burns because of their relation to overall prognosis.

a. Area of the burns; expressed in percentage of body surface involved.

b. Involvement of critical organs; i.e., head and neck, respiratory tract, genitalia, hands, and feet.

c. Depth of burn; superficial (first- or second-degree), or deep (second degree) and full thickness (third degree).

607. Area of Burn.

a. The most accurate way to estimate the amount of tissue injury following a burn is to measure the extent of the body surface burned. However, direct measurement is not generally possible or necessary, and a short cut method of estimating the percent of the body surface involved can be very useful. The "Rule of Nines" method is a simple and reasonably reliable guide in which the various parts of the body are divided into surface areas of 9% each (or multiples of 9%) as shown in Table 6-I.

Table 6-I. Rule of Nines for Establishing Extent of Body Surface Burned

Anatomic surface	% of total surface
Head and neck	9 = 9
Anterior trunk	2 x 9 = 18
Posterior trunk	2 x 9 = 18
Upper limbs	9 ea = 18
Lower limbs	18 ea = 36
Genitalia and perineum	1 = 1

b. As the percent of the surface burned increases, morbidity and the probability of mortality increases sharply. Burns which cover 20% or more of the body surface can be fatal without treatment. Even with treatment, mortality from extensive burns will be high, particularly in the very young or the aged. Young healthy soldiers who have uncomplicated burns may survive even extensive involvement with proper care.

c. Determination of the percent of the body involved will aid in planning resuscitative treatment and estimating fluid requirements during the first 48 hours after the burn injury. Patients with severe burns will suffer quite extensive fluid and electrolyte

losses, resulting in severe hypovolemic shock requiring aggressive fluid replacement therapy as early as possible. An outline of a resuscitative program is given in the treatment section.

608. Involvement of Critical Organs.

When certain organ systems are involved, the clinical effects of burns can be quite serious in spite of the fact that only a small fraction of the body is involved.

a. *Head and Neck Burns*. Burns of the face can be serious problems, even if the eyes are not involved. Burns of the head frequently are complicated by severe edema, which can result in respiratory obstruction. This can be quite serious when the inhalation of hot gases has occurred. It may be necessary to do tracheotomies on many of these patients.

b. *Burns of the Respiratory Tract*. When hot gasses are inhaled, this very serious type of injury may be sustained. These injuries have a high probability of mortality if the burns extend deep into the alveoli. These patients are very fragile and may not tolerate early evacuation. Pulmonary edema may develop abruptly, without warning, requiring vigorous ventilator support. These injuries can be very difficult to manage.

c. *Burns of Hands and Feet*. These can be very disabling and may require long hospitalization for extensive surgical care even though they are not life threatening injuries. These patients may not be able to care for themselves and, as a result, will require extensive nursing care.

609. Depth of Burn.

Burns are classified on the basis of the depth of the injury.

a. *Superficial or Partial Skin Thickness Burns*. These are lesions in which the dermis is intact and only the epidermis is injured. When the injury is limited and only erythema occurs (such as in a sunburn), these are usually called first-degree burns. If blistering is seen, the injuries are called second-degree burns. Superficial burns are usually painful but will heal readily by epithelization unless infection occurs. Infection can convert a typical second-degree, superficial burn into a deep or full-thickness burn which will not heal by epithelization but rather by scarring. Second-degree burns will be very common in nuclear combat and may be the one most common injury seen.

b. *Deep or Full-Thickness Burns*. Injuries involving the full thickness of the skin which cannot heal by epithelization are called third-degree burns. Instead, these

injuries heal by scarring, and as a result there may be contraction and loss of function, particularly if extremities are involved. Extensive plastic surgery may be required to prevent or limit loss of function. The areas of a burn which are third-degree are usually painless, and this helps differentiate areas of third from second-degree when both are present. The earlier the diagnosis of the degree of burn is made, the sooner reconstructive treatment with skin grafting can be started. In general, however, in nuclear combat, early skin grafting will rarely be possible.

610. Treatment.

Initial treatment of burn patients will be resuscitative. When such patients are first seen, a simple plan of treatment must include: maintenance of airway with ventilating support as needed, adequate fluid therapy, and careful records of input and output.

a. *Maintenance of Airway*. This is of particular importance in head and neck burns or in unconscious patients. If large numbers of patients are seen requiring transportation over long distances early in the postburn period, tracheotomies may have to be done on a routine basis. Tracheotomies done prior to the onset of edema are much easier to perform than when they are done after edema has resulted in respiratory obstruction. When only small numbers of patients require treatment, tracheotomies are rarely required.

b. *Fluid Therapy*. The shock that is associated with an extensive burn will be severe, and survival of these patients depends upon adequate, balanced fluid replacement therapy. In combat, however, standardized methods of management are required. Standard formulae for determining the fluid requirements of burn patients have been developed and can be used in combat. The basic principle in these formulae is that the amount of fluid required is proportional to the percent of body surface burned and body weight. The type of fluid used includes colloidal materials to replace the plasma constituents lost as well as electrolytes.

c. *Fluid Requirements for First 24 Hours.*

(1) Colloid solutions: 0.5 ml x body weight in kilos x percent of body surface burned.

(2) Electrolyte solutions: 1.5 x body weight in kilos x percent of body surface burned.

(3) Additional fluids: 2000 ml 5-10% dextran in water.

d. *Example*. This formula, to meet the requirements of a 70-kg person with 30% body surface burn, would be:

Colloid:	0.5 ml x 70 x 30	=	1050 ml
Electrolyte:	1.5 ml x 70 x 30	=	3150 ml
Metabolic fluid (carbohydrates):			2000 ml
Total:			6200 ml

e. *Restrictions*. Certain restrictions on the application of this formula are required since it is only a guide.

(1) Fluid requirements for an injury involving more than 50% of the body surface should be calculated as if the burn were no more than 50%.

(2) 10,000 ml of fluid should be the maximum given in the first 24 hours.

(3) The first half of the fluid should be given more rapidly than the second; and the actual rate of administration should be 0adjusted according to urinary output.

(4) During the second 24 hours, the colloid and electrolyte given should be about one-half of that given during the first 24 hours. Again, the actual rate should be adjusted to maintain a reasonable urinary output. This is the single best clinical guide to use in determining the patient's actual fluid requirements.

(5) After the 3rd or 4th day, the patients will begin to resorb fluid from the edematous areas and will excrete it in large quantities. Administration of fluids to replace this loss is contraindicated. Excessive administration of fluids must be avoided during this time, and fluid intake can generally be reduced to that normally required for metabolic needs.

f. *Input and Output Records*. It is extremely important to accurately follow the input and output of fluids in burn patients. It would be impossible to modify fluid therapy according to individual needs without accurate records. Combat medical records, however, must be simple and should be attached to the patient so that they accompany him during evacuation. Medical planners must consider how to modify and improve combat medical records so that accurate input and output data on burn patients can be recorded. Most burn patients will require urinary catheterization, and this can aid considerably in recording urinary output rates accurately.

611. Care of Burn Wound.

Although first priority in patient care is resuscitation, proper care of the burn wound is essential both for survival as well as for optimum healing and preservation of function. In that regard, as soon as the patient's overall condition permits, after hospitalization, initial debridement and cleaning of the burn should be done. The main purpose of this treatment is to remove foreign material and dead tissue to minimize infection. Thorough irrigation and the application of topical antimicrobial creams such as argentic sulfadiazine and sterile dressings should complete the initial procedures. Special attention should be given to critical areas such as the hands and surfaces over joints.

SECTION IV - RADIATION INJURY AND COMBINED INJURY

612. General.

Radiation injury alone or in conjunction with other injuries or diseases will be common in nuclear warfare. Radiation injury can result from a single exposure to prompt radiation at the time of detonation of a nuclear weapon, from exposure to high levels of fallout radiation, or from repeated exposures to both with complex patterns of recovery from an accumulation of radiation damage.

a. Whole-body irradiation, where absorbed doses are high and acquired over short periods of time, will result in acute radiation sickness. There are three characteristic syndromes which make up the typical clinical pattern of acute radiation sickness. These are the hematopoietic, gastrointestinal, and neurovascular syndromes which occur with increasing dose respectively.

b. The hematopoietic syndrome, or syndrome of bone-marrow depression, occurs at lower doses than the others and would be the most common form of radiation sickness seen in nuclear combat. Manifestations of bone-marrow depression are seen following doses of radiation in the low through midlethal range. As the probability of lethality becomes 100 percent with higher doses, the gastrointestinal syndrome will predominate. This syndrome, which will also be common, develops from combined severe damage to bone marrow and the gastrointestinal tract. The neurovascular syndrome is associated with absorbed doses in the supralethal range and would be seen quite rarely since heat and blast effects would cause immediate lethality in most situations where the required very high radiation doses would be sustained. Exceptions could occur in aircrews exposed to prompt nuclear radiation from high altitude detonations and personnel protected against heat and blast in hardened sites below the surface or personnel in vehicles or shelters in the proximity of enhanced weapons' detonations. In these circumstances, an increase in the numbers of casualties receiving radiation doses in the supralethal range can be expected.

613. Clinical Course of Radiation Sickness.

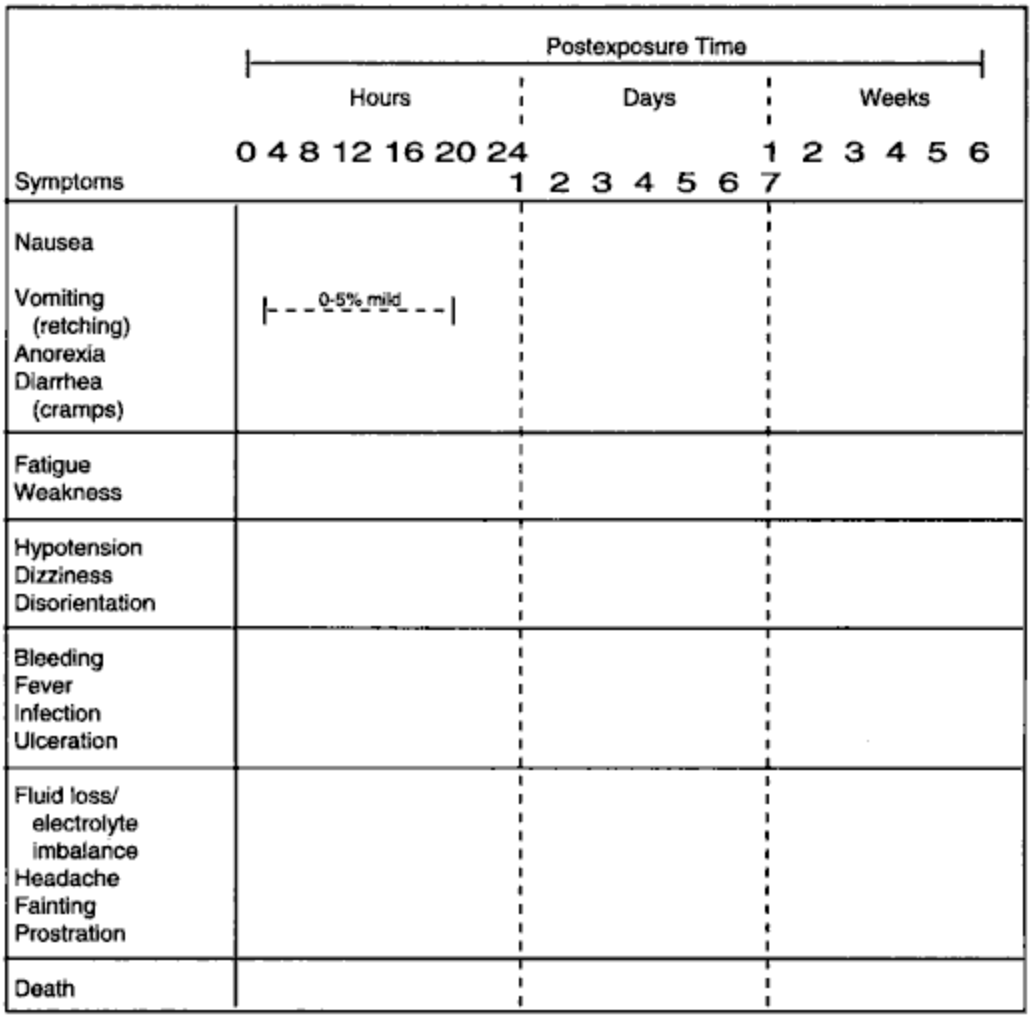
The three syndromes described follow a similar clinical pattern that can be divided into three phases: an initial or prodromal phase occurring during the first few hours after exposure; a latent phase, which becomes shorter with increasing dose; and the manifest phase of clinical illness. The time of onset and degree of the transient incapacitation of the initial phase, the duration of the latent period, as well as the time of onset and severity of the clinical phase and ultimate outcome are all to a variable extent, dose dependent.

a. *Prodromal Phase*. The initial phase of prodromal symptoms is characterized by the relatively rapid onset of nausea, vomiting, and malaise. This is a nonspecific clinical response to acute radiation exposure. It is not diagnostic of the degree of radiation injury; however, in the absence of associated trauma and an early onset, it does suggest a large radiation exposure. This radiogenic vomiting should not be confused with psychogenic vomiting which results from stimulation of the central nervous system by the sight/odor of blood, mutilation, vomitus, or excrement. The duration of this prodromal phase is short, generally a few hours, and the incapacitation should not be severe enough to warrant evacuation of military personnel away from their units.

b. *Latent Phase*. Following recovery from the prodromal phase, there will be a latent phase during which the exposed individual will be relatively symptom-free. The length of this phase varies with the dose and the nature of the later clinical phase. The latent phase is longest preceding the bone-marrow depression of the hematopoietic syndrome and may vary between 2 and 6 weeks. It is somewhat shorter prior to the gastrointestinal syndrome, lasting from a few days to a week. It is shortest of all preceding the neurovascular syndrome, lasting only a matter of hours. These times are exceedingly variable and may be modified by the presence of other disease or injury. Because of the extreme variability, it is not practical to hospitalize all personnel suspected of having radiation injury early in the latent phase unless radiation injury has reliably been diagnosed. Instead, it is much more reasonable to wait until the onset of the phase of clinical illness or the development of significant hematopoietic suppression as indicated by the individual's peripheral blood profile.

c. *Manifest Phase*. This phase presents with the clinical symptoms associated with the major organ system injured (marrow, intestine, neurovascular system). A summary of essential features of each syndrome and the doses at which they would be seen in young healthy adults exposed to short, high dose single exposures is shown in Figure 6-I. The details of the clinical courses of each of the three syndromes are subsequently described.

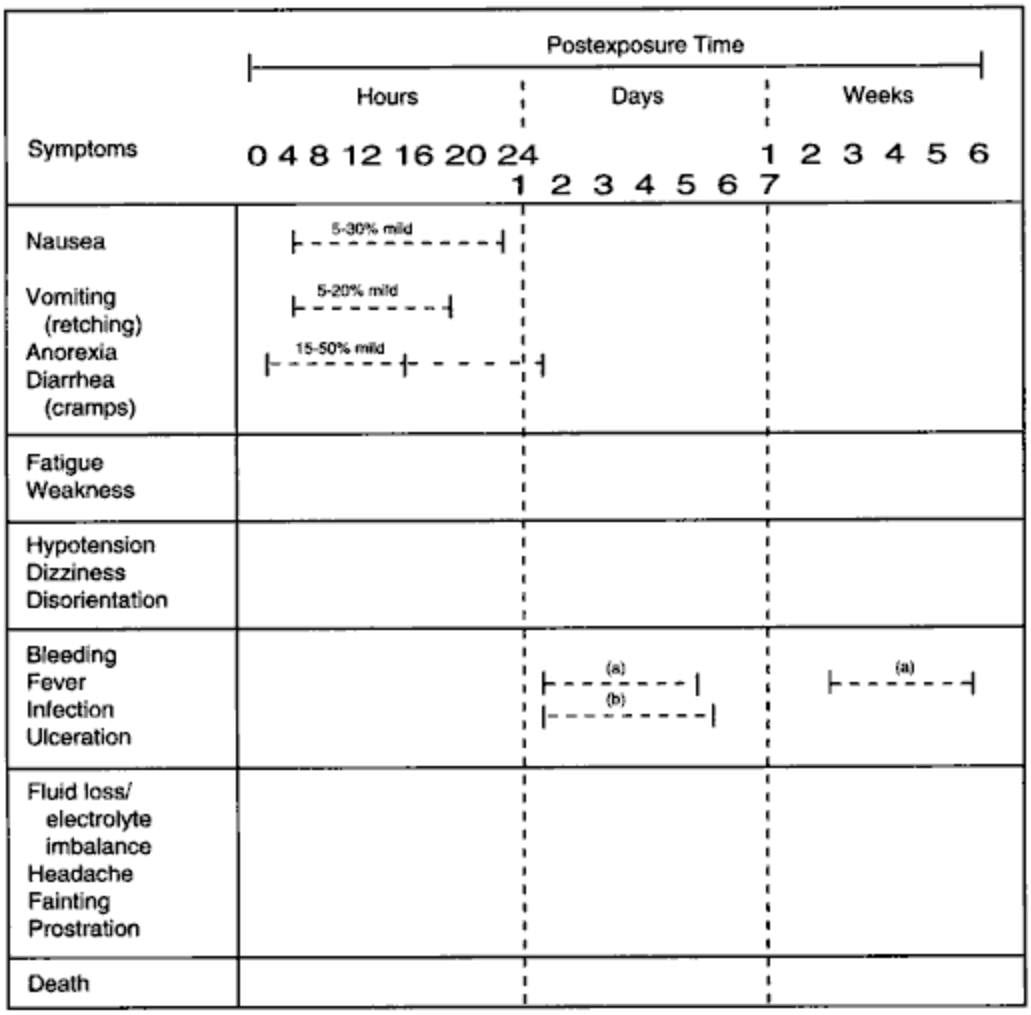
Symptoms for Dose Range 0 to 75 cGy Free in Air



Management and Treatment		
Performance:	Hospitalization Percentage/ Duration:	Therapy:
Combat Effective.	None.	None.

Figure 6-1. Acute Clinical Effects of Single-Dose Rate Exposure of Whole-Body Irradiation to Healthy Adults (1 of 9)

Symptoms for Dose Range 75 to 150 cGy in Free Air

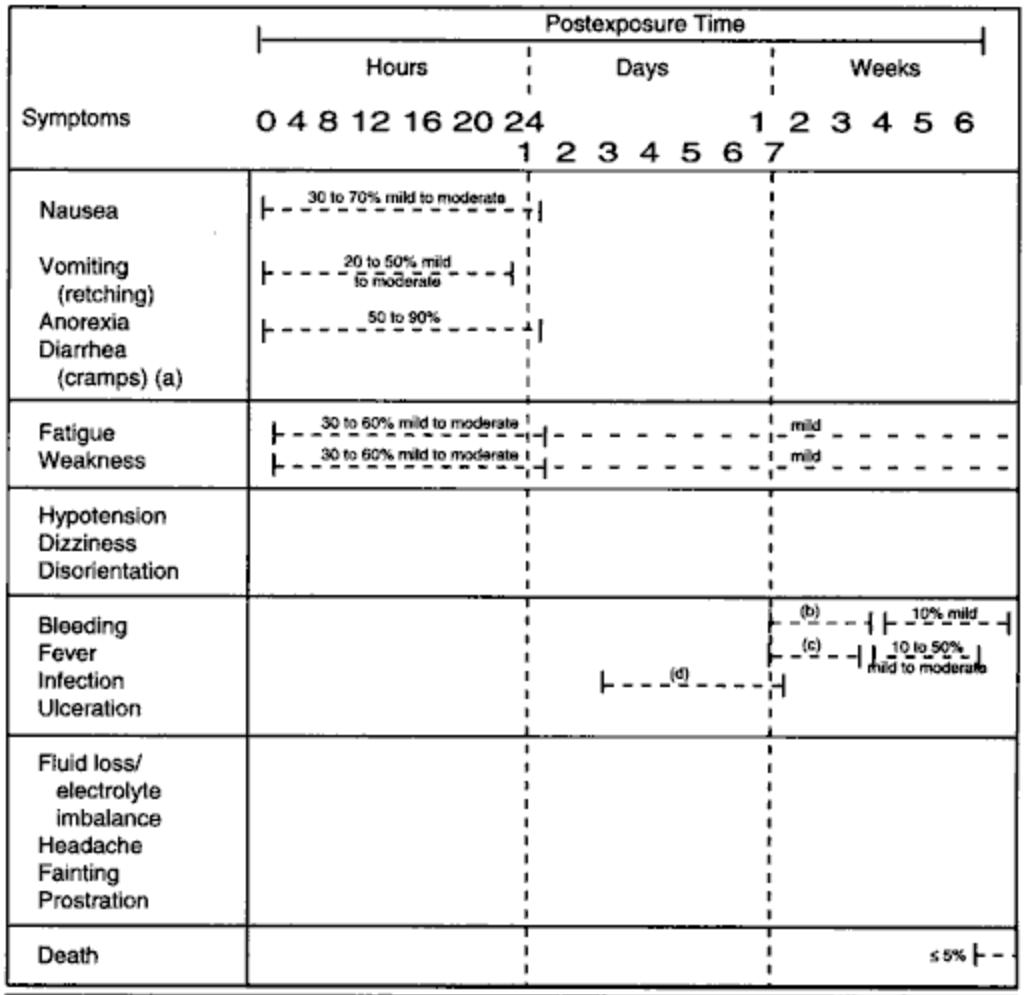


Management and Treatment		
Performance:	Hospitalization Percentage/ Duration:	Therapy:
Combat Effective.	None.	None.

(a) Slight drop in lymphocyte, platelet, and granulocyte counts.
(b) Increased susceptibility to non-opportunistic pathogens.

Figure 6-I. Acute Clinical Effects of Single-Dose Rate Exposure of Whole-Body Irradiation to Healthy Adults (2 of 9)

Symptoms for Dose Range 150 to 300 cGy in Free Air



Management and Treatment		
Performance: DT:PD from 4 hours until recovery. UT:PD from 6 to 20 hours and from 6 weeks until recovery.	Hospitalization Percentage/ Duration: At 3 to 5 weeks medical care for 10 to 50 percent; survivors return to duty.	Therapy: Symptomatic treatment with antiemetics and antibiotics; hematologic surveillance.

(a) Ten percent of the Marshallese victims exposed to 175 rads (cGy) experienced diarrhea during the first exposure day.
(b) Slight to moderate drop in platelets: from 3 x 10^5/mm^3 to 1.8-0.8 x10^5/mm^3.
(c) Slight to moderate drop in granulocytes: from 6 x 10^3/mm^3 to 4.5-2.0 x 10^3/mm^3.
(d) Slight to moderate drop in lymphocytes: from 3 x 10^3/mm^3 to 2.0-1.0 x 10^3/mm^3. Susceptibility to opportunistic pathogens.

CI = Combat Ineffective (less than 25% performance)
PD = Performance Degraded (25 to 75% performance)
DT = Demanding Task
UT = Undemanding Task

Figure 6-I. Acute Clinical Effects of Single-Dose Rate Exposure of Whole-Body Irradiation to Healthy Adults (3 of 9)

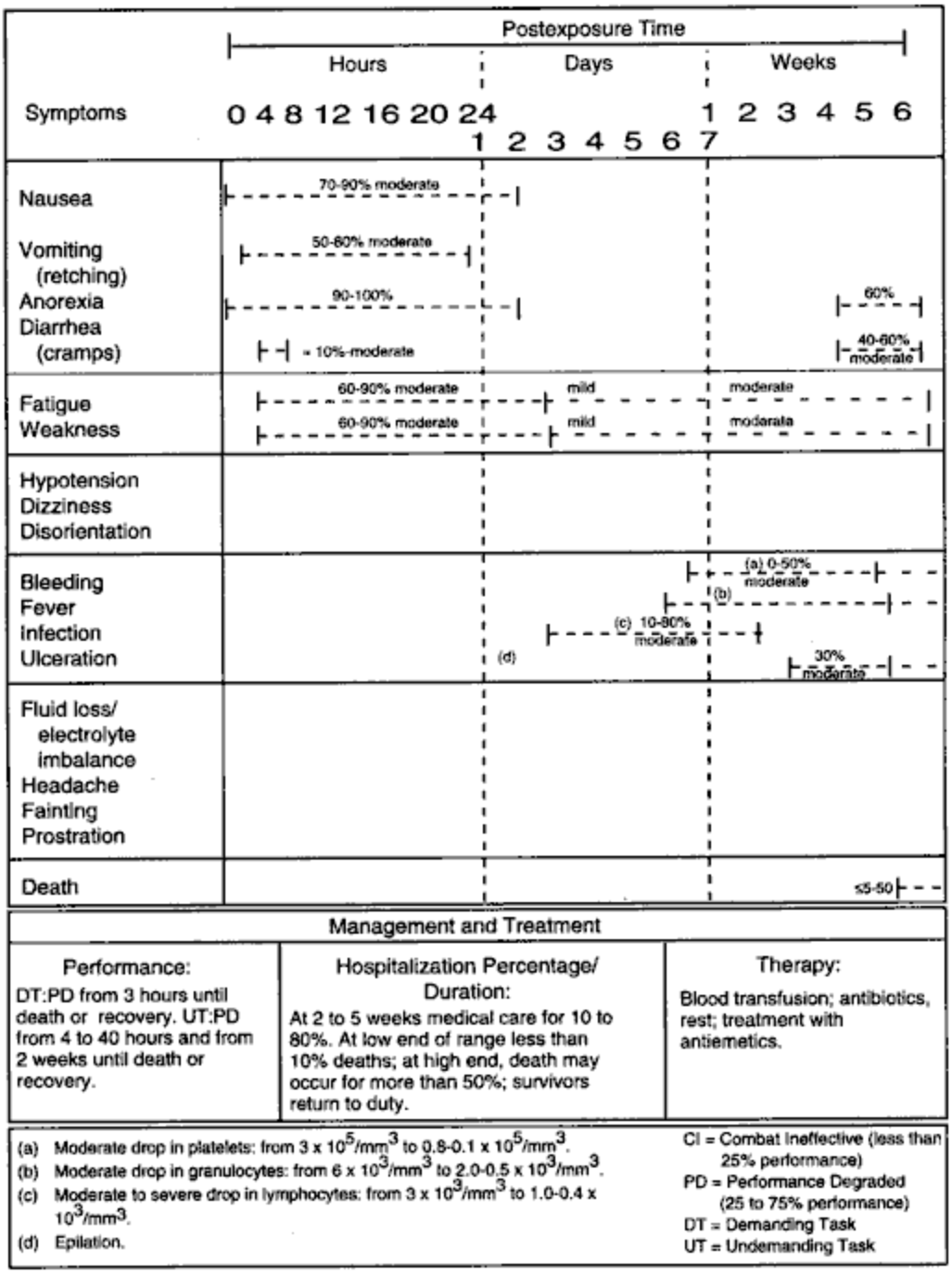


Figure 6-I. Acute Clinical Effects of Single-Dose Rate Exposure of Whole-Body Irradiation to Healthy Adults (4 of 9)

Symptoms for Dose Range 530 to 830 cGy in Free Air

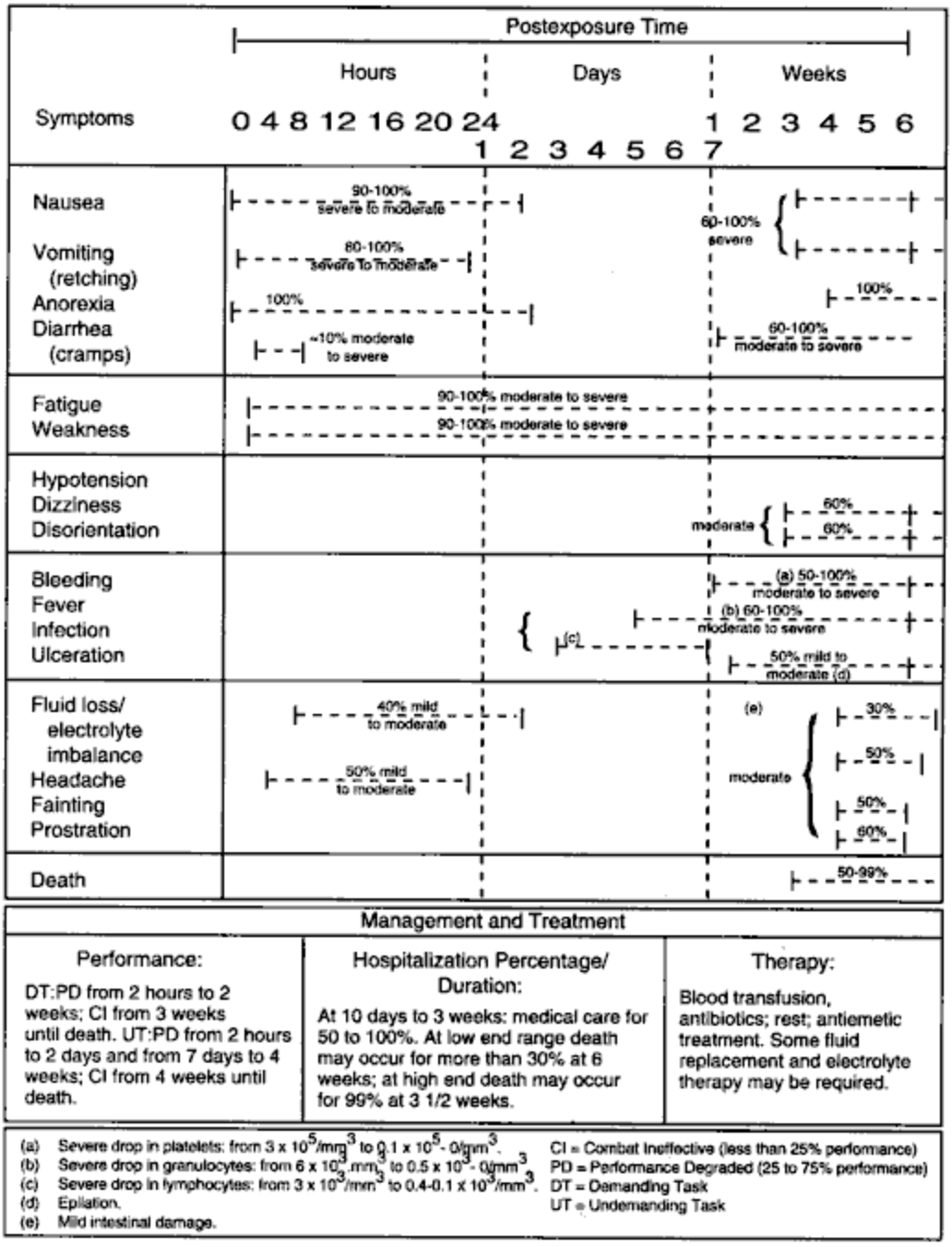
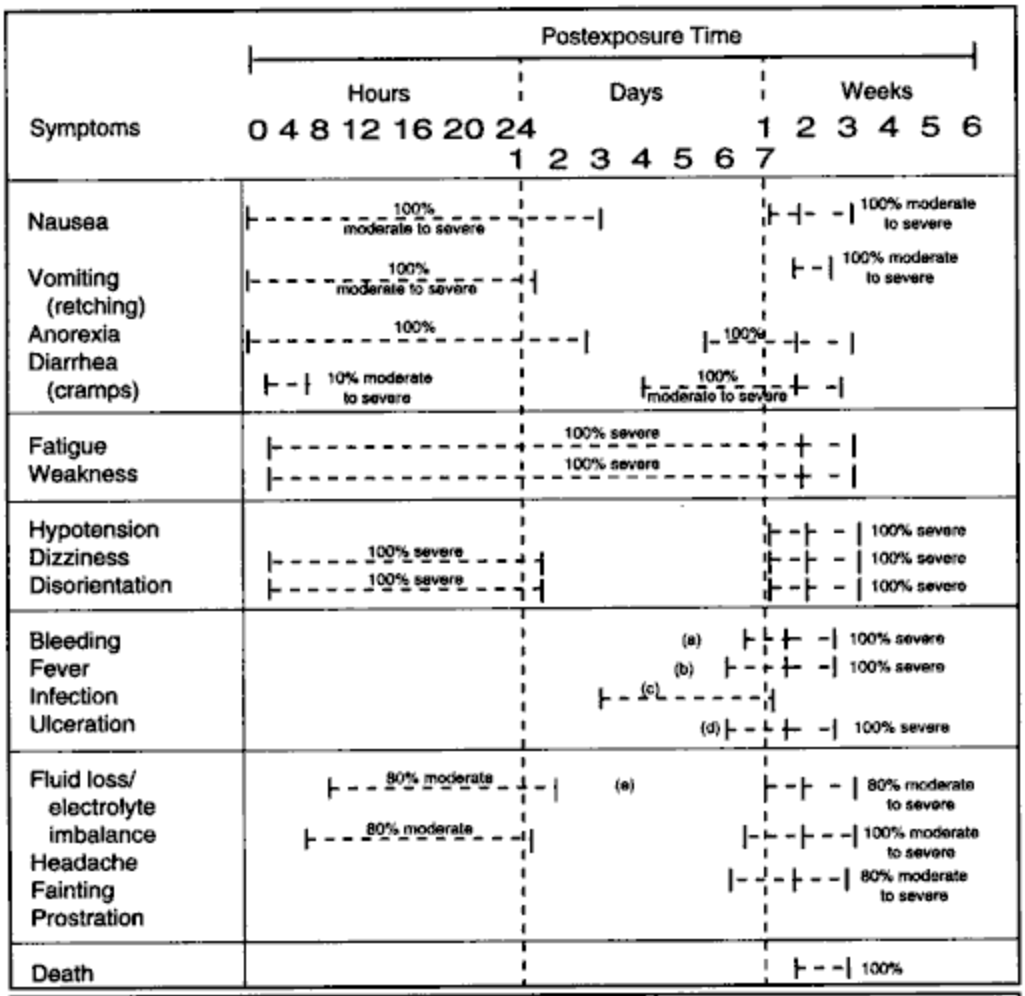


Figure 6-1. Acute Clinical Effects of Single-Dose Rate Exposure of Whole-Body Irradiation to Healthy Adults (5 of 9)

Symptoms for Dose Range 830 to 1100 cGy in Free Air



Management and Treatment		
Performance: DT:PD from 1.5 to 8 hours; CI from 1 hour to 1.2 days; PD 1.2 to 10.4 days to death. UT:PD 2 hours to 3 days. Combat effective 3 to 4 days: PD from 4 to 14 days; CI from 14 days to death.	**Hospitalization Percentage/ Duration:** 4 to 6 days medical care for 100%; 100 % deaths at 2 to 4 weeks.	**Therapy:** Maintenance of electrolyte balance plus treatment recommended for lower doses.

(a) Platelet count drops nearly to zero.
(b) Granulocyte count drops nearly to zero.
(c) Lymphocyte count drops nearly to zero.
(d) Epilation.
(e) Moderate intestinal damage.

CI = Combat Ineffective (less than 25% performance)
PD = Performance Degraded (25 to 75% performance)
DT = Demanding Task
UT = Undemanding Task

Figure 6-1. Acute Clinical Effects of Single-Dose Rate Exposure of Whole-Body Irradiation to Healthy Adults (6 of 9)

Symptoms for Dose Range 1100 to 1500 cGy in Free Air

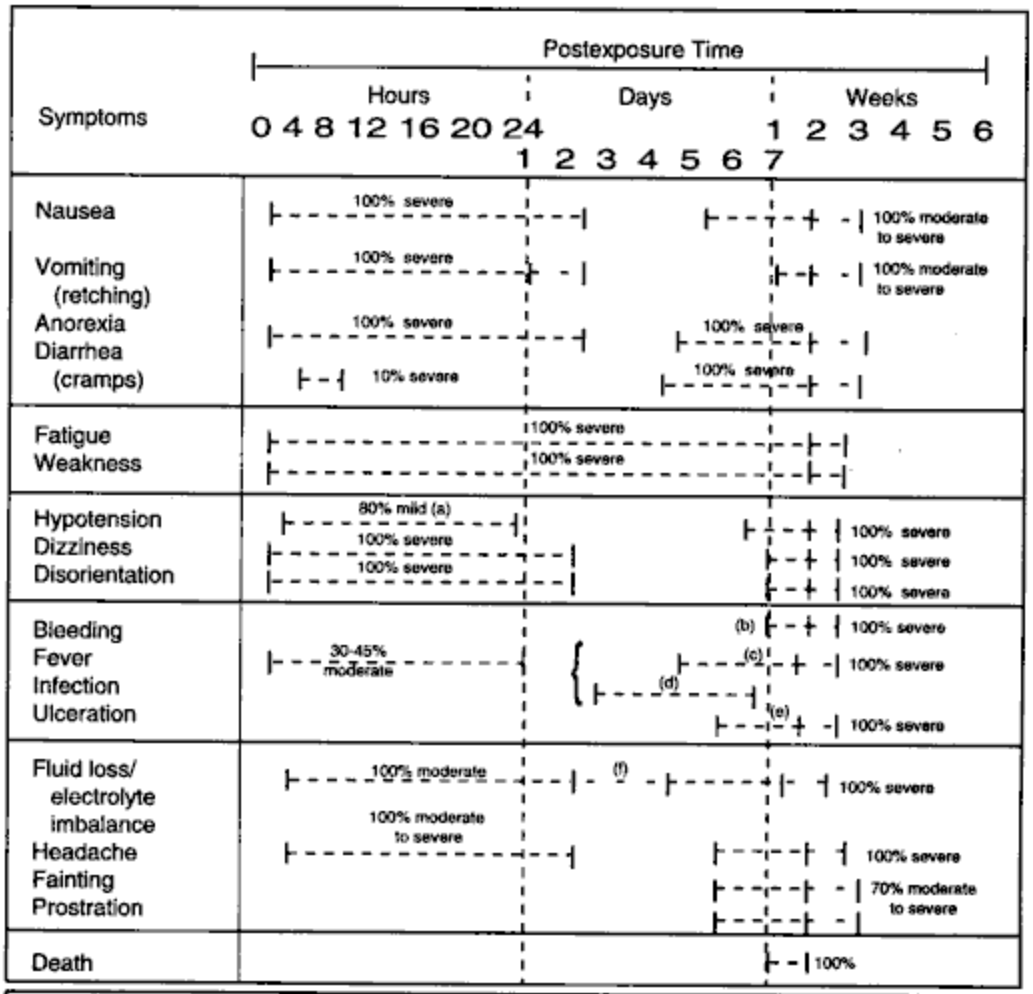


Figure 6-1. Acute Clinical Effects of Single-Dose Rate Exposure of Whole-Body Irradiation to Healthy Adults (7 of 9)

Symptoms for Dose Range 1500 to 3000 cGy in Free Air

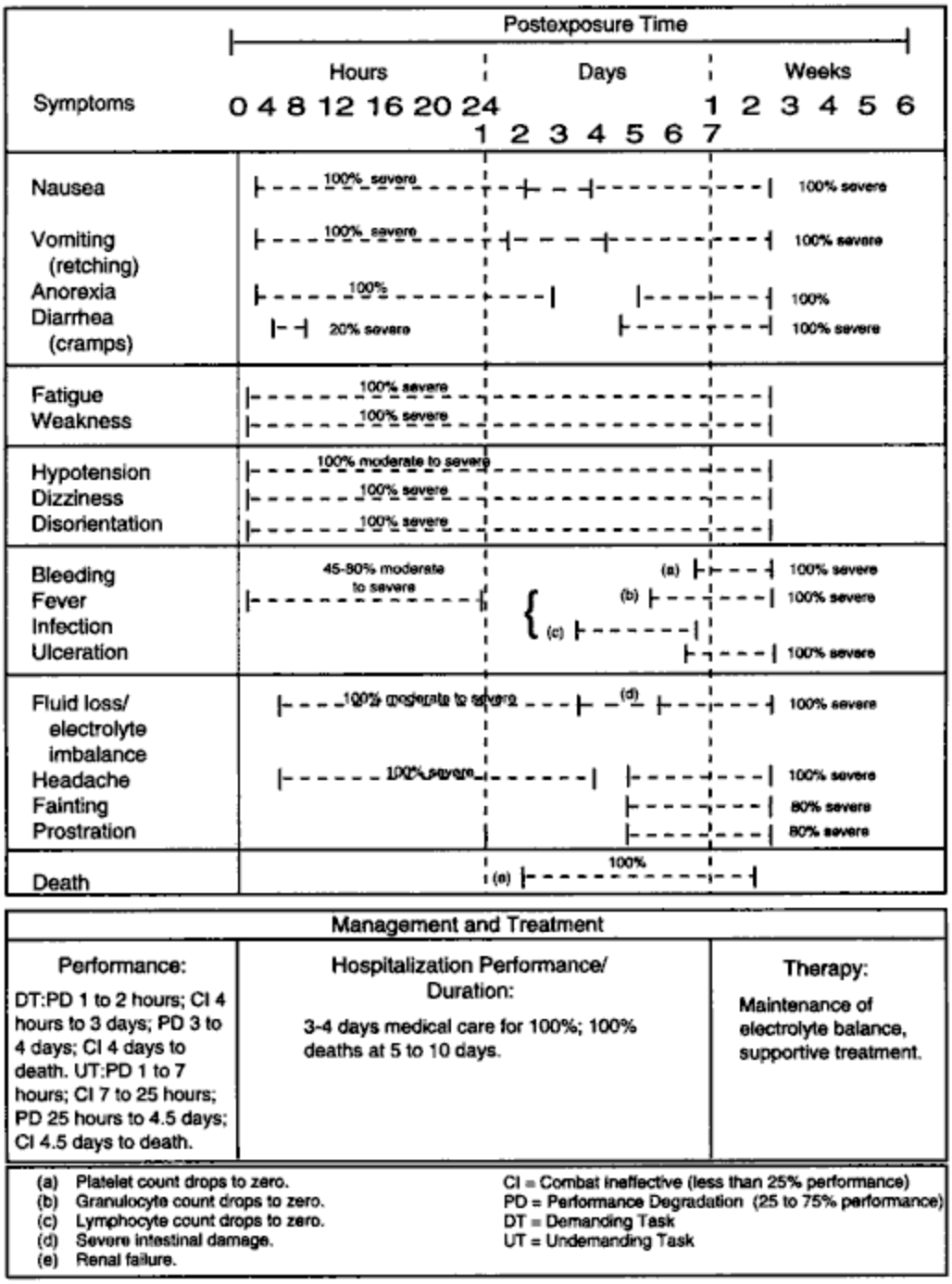


Figure 6-I. Acute Clinical Effects of Single-Dose Rate Exposure of Whole-Body Irradiation to Healthy Adults (8 of 9)

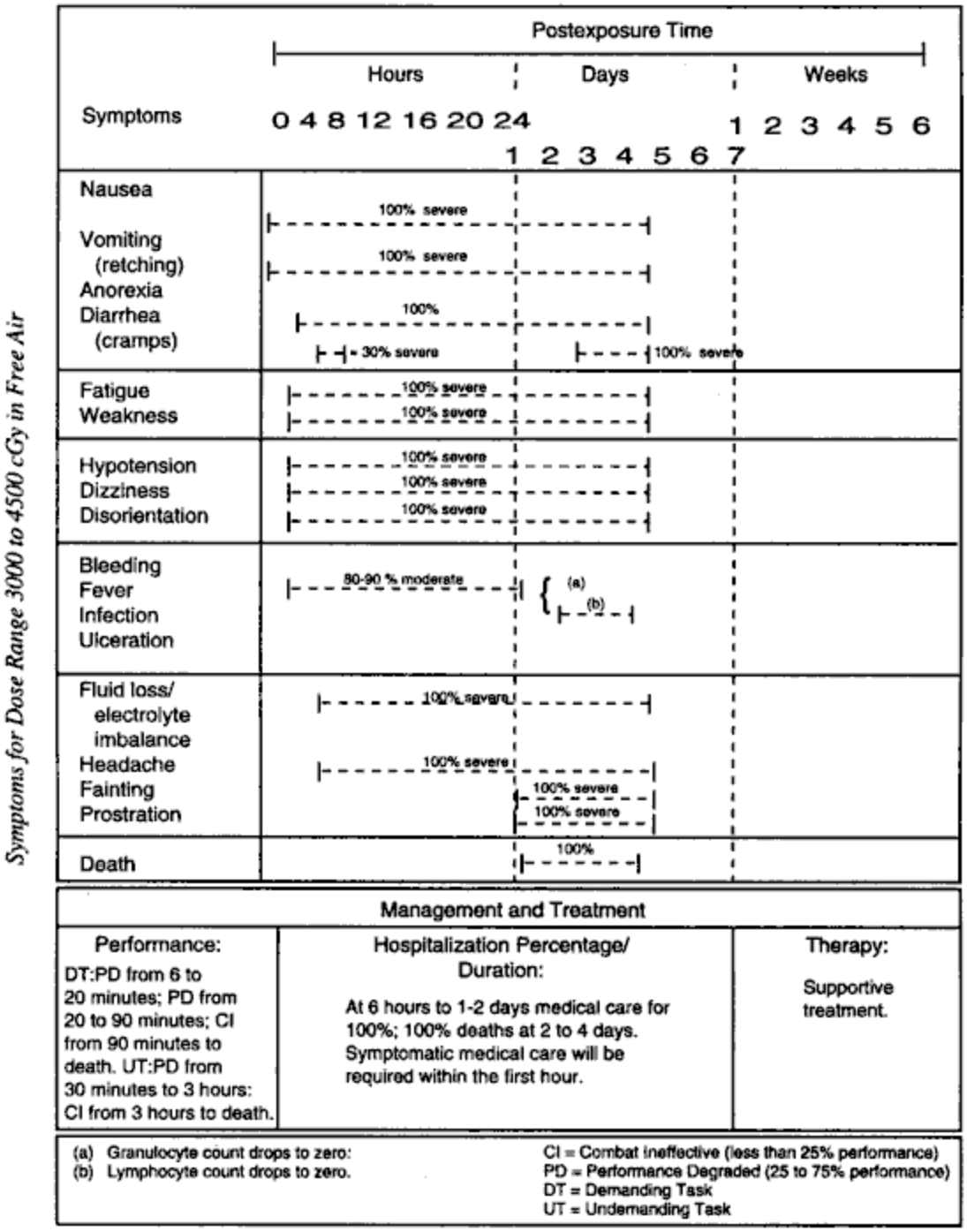


Figure 6-I. Acute Clinical Effects of Single-Dose Rate Exposure of Whole-Body Irradiation to Healthy Adults (9 of 9)

614. Hematopoietic Syndrome.

a. Patients who have received doses of radiation in the low to midlethal range will have depression of bone-marrow function with cessation of blood-cell production leading to pancytopenia. Changes within the peripheral blood profile will occur as early as 24 hours post irradiation. The exact time sequence of the depression of various circulating cell lines will vary. Lymphocytes will be depressed most rapidly and erythrocytes least rapidly. Other leukocytes and thrombocytes will be depressed somewhat less rapidly than lymphocytes. The time of onset of the depression of cellular production in the marrow will vary considerably, and the concomitant clinical problems of a tendency toward uncontrolled hemorrhage, decreased resistance to infection, and anemia will likewise vary considerably from as early as 10 days to as much as 6 to 8 weeks after exposure.

b. A reasonable average time of onset of clinical problems of bleeding and anemia and decreased resistance to infection is 2 to 3 weeks. In general, the severity of the hematological depression will be dose dependent, and the more severe phases of bone-marrow depression will occur earlier. However, even lethal cases of bone-marrow depression may not occur until 6 weeks after exposure. The presence of other injuries will increase the severity and accelerate the time of maximum bone-marrow depression

c. If the exposures leading to the bone-marrow depression are multiple, the time of onset of depression will be very difficult to estimate. The clinical picture, however, once bone-marrow depression is present, will be identical regardless of the sequence of exposure.

d. The most useful laboratory procedure to evaluate bone-marrow depression is the peripheral blood count. A pancytopenia with particularly severe depression of lymphocytes, granulocytes, and thrombocyte will be strongly indicative of radiationinduced bone-marrow depression. (See Figures 6-II, 6-III, and 6-IV.) Bone-marrow studies will rarely be possible under field conditions and will add little information to that which can be obtained from a careful peripheral blood count.

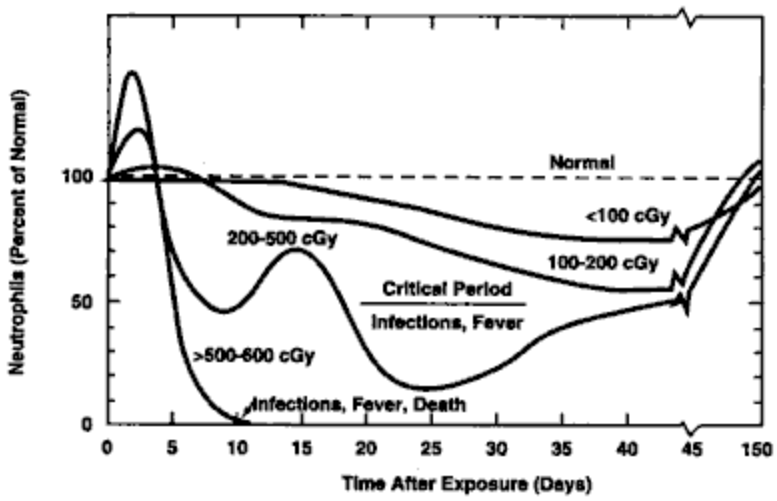


Figure 6-II. Smoothed Average Time-Course of Neutrophil Changes in Human Cases from Accidental Exposure as a Function of Dose

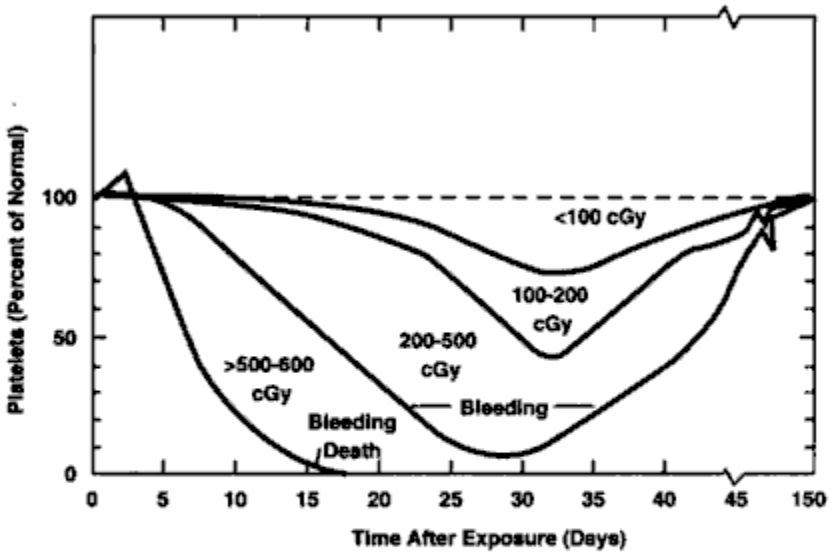


Figure 6-III. Smoothed Average Time-Course of Platelet Changes in Human Cases from Accidental Radiation Exposure as a Function of Dose

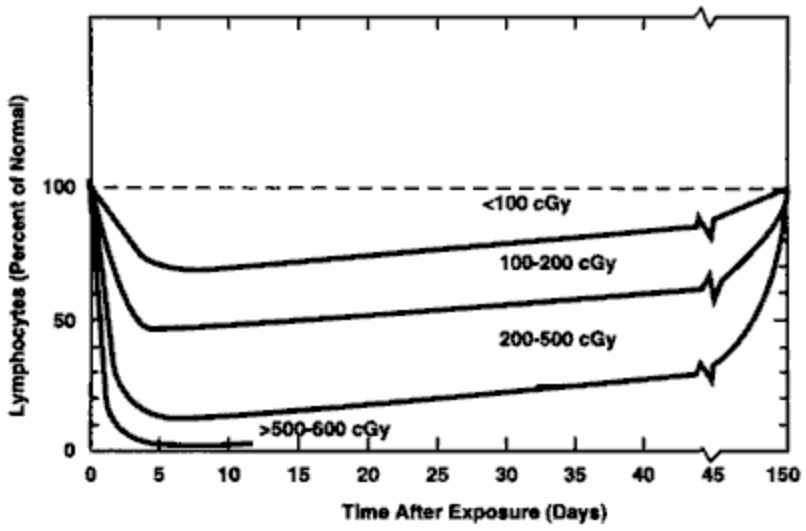


Figure 6-IV. Smoothed Average Time-Course of Lymphocyte Changes in Human Cases from Accidental Radiation Exposure as a Function of Dose

e. Patients will show signs or increased evidence of hemorrhagic disease and increased susceptibility to infection. If an infection occurs, there may be little clinical response because of the concomitantly depressed inflammatory response. The patients will lose weight, may lose hair and ultimately die from overwhelming infection and hemorrhage unless sufficient regeneration of the marrow occurs. Following lethal exposures, the marrow may be so damaged that recovery will be impossible.

615. Gastrointestinal Syndrome.

a. The doses of radiation which will result in the gastrointestinal syndrome are higher than those causing the hematopoietic syndrome. An acute dose which will cause this syndrome would be at least 800 cGy measured in air. Under certain circumstances, lower doses may cause this syndrome, and conversely, exposures to high doses at low dose rates or as fractionated exposures may not cause it. Regardless of the dose involved, the gastrointestinal syndrome has a very serious prognosis because it is almost always accompanied by nonrecoverable bone-marrow.

b. The onset of the clinical phase of the gastrointestinal syndrome occurs earlier than that of the hematopoietic syndrome. After a short latent period of a few days to a week or so, the characteristic severe fluid losses, hemorrhage and diarrhea begin. The pathologic basis for this syndrome is an early physiologic derangement of the epithelial cells followed by a combination of severe loss of intestinal mucosa and injury to the fine vasculature of the submucosa. There is no specific clinical sign which is pathognomonic of radiation-caused gastrointestinal damage. However, a

peripheral blood count done on these patients should show an early onset of a severe pancytopenia occurring as a result of the bone-marrow depression.

c. A problem in diagnosis will arise in patients with sublethal hematopoietic depression due to radiation and diarrhea due to some other cause such as infection. It would be difficult to differentiate patients with lethal radiation sickness from those with potentially nonlethal radiation sickness complicated by dysentery. Microscopic examination of the diarrhea may reveal inflammatory cells which is suggestive of dysentery. Radiation enteropathy is not likely to result in an inflammatory response. It must be assumed during the care of all patients that even those with a typical gastrointestinal syndrome may be salvageable, until blood counts indicate that the bone-marrow depression is irreversible.

616. Neurovascular Syndrome.

This syndrome is associated only with very high acute doses of radiation. The lower limit is probably 2000 to 3000 cGy, although hypotension (significant decline in systemic blood pressure) may be seen at even lower doses. The latent period is very short varying from several hours to 1 to 3 days. The subsequent clinical picture is basically that of a steadily deteriorating state of consciousness with eventual coma and death. Convulsions may or may not occur. There may be little or no indication of increased intracranial pressure. Because of the very high doses of radiation required to cause this syndrome, personnel close enough to a nuclear explosion to receive such high doses would generally be well within the range of 100% lethality due to blast and thermal effects. However, in nuclear detonations above the atmosphere with essentially no blast, very high fluxes of ionizing radiation may extend out far enough to result in high radiation doses to aircraft crews. Such personnel could conceivably manifest this syndrome, uncomplicated by blast or thermal injury. Personnel protected from blast and thermal effects in shielded areas could also sustain such doses. Still, very few patients will be hospitalized with this syndrome.

617. Diagnosis.

a. The diagnosis of radiation sickness is based primarily upon the clinical picture presented by the patient. A precise history of exposure may be very difficult to obtain, since many individuals may not know that they actually have been exposed to radiation, particularly if the exposure is due to fallout. The physical findings and characteristics of the various forms of radiation sickness are described below, along with such laboratory findings as may occur. Dosimetry, at the present time, will not give adequate information to determine either the extent of radiation injury or the prognosis. Dosimeters cannot tell whether a radiation exposure is whole body or partial body. They do not tell what the dose rate of the exposure was. Finally, they

cannot differentiate between single exposures and multiple exposures unless read at regular intervals.

b. These unknowns, coupled with the marked effects of age or physical condition, of concomitant disease, and of stress, etc., make it essential that physicians with the responsibility for treating patients in a hospital, base their treatment decisions primarily upon the actual clinical condition of the patient. However, in the mass casualty situation, decisions based on dosimetric data alone may be all that is practicable.

c. Consequently, the following guidelines based on recent recommendations apply to medical personnel operating in austere field conditions. Lymphocyte levels may be used as a biologic dosimeter to confirm the presence of pure radiation injury but not in combined injuries. If the physician has the resources of a clinical laboratory, additional information can be obtained to support the original working diagnosis by the presence of prodromal symptoms. An initial blood sample for concentrations of circulating lymphocytes should be obtained as soon as possible from any patient classified as "Radiation Injury Possible" or "Radiation Injury Probable." After the initial assessment or at least no later than 24 hours after the event in question, additional blood samples should be taken for comparison. The samples may be interpreted as follows:

(1) *Lymphocyte levels in excess of 1500/mm^3 (cubic millimeters).* The patient most likely has not received a significant dose that would require treatment.

(2) *Lymphocyte levels between 1000 and 1500/mm^3.* The patient may require treatment for moderate depression in granulocytes and platelets within 3 weeks postexposure.

(3) *Lymphocyte levels between 500 and 1000/mm^3.* The patient will require treatment for severe radiation injury. The patient should be hospitalized to minimize the complications from hemorrhage and infection that will arise within 2-3 weeks postexposure.

(4) *Lymphocyte levels of less than 500/mm^3.* The patient has received a radiation dose that may prove fatal. The patient needs to be hospitalized for the inevitable pancytopenic complications.

(5) *Lymphocytes not detectable.* The patient has received a superlethal radiation dose, and survival is very unlikely. Most of these patients have received severe injuries to their gastrointestinal and cardiovascular systems and will not survive for more than 2 weeks.

(6) *Other Guidelines.* A useful rule of thumb is, if lymphocytes have decreased by 50% and are less than 1000/mm^3, then the patient has received a significant radiation exposure. In the event of combined injuries, the use of lymphocytes may be unreliable. Patients who have received severe burns or multisystem trauma often develop lymphopenia.

d. It is difficult to establish an early definitive diagnosis. Therefore, it is best to function within a simplified, tentative classification system based on the three possible categories of patients noted in Table 6-II and discussed in the following.

Table 6-II. Preliminary Triage of Casualties with Possible Radiation Injuries

	Possible category of radiation injury		
Symptoms	Unlikely	Probable	Severe
Nausea	-	++	+++
Vomiting	-	+	+++
Diarrhea	-	±	± to +++
Hyperthermia	-	±	+ to +++
Hypothermia	-	-	+ to ++
Erythema	-	-	- to ++
CNS dysfunction	-	-	- to ++

- = Absent
+ = Present
++ = Excessive
+++ = Very Excessive

(1) *Radiation Injury Unlikely.* If there are no symptoms associated with radiation injury, patients are judged to be at minimal risk for radiation complications. These patients should be triaged according to the severity of the conventional injuries. If the patients are free of conventional injuries or disease states that require treatment, they should be released and returned to duty.

(2) *Radiation Injury Probable.* Anorexia, nausea, and vomiting are the primary prodromal symptoms associated with radiation injury. Priority for further evaluation will be assigned after all life-threatening injuries have been stabilized. Casualties in this category will not require any medical treatment

within the first few days for their radiation injuries. Evidence to support the diagnosis of significant radiation injury in the absence of burns and trauma may be obtained from lymphocyte assays taken over the next 2 days. If the evidence indicates that a significant radiation injury was received, these casualties need to be monitored for pancytopenic complications.

(3) *Radiation Injury Severe*. These casualties are judged to have received a radiation dose that is potentially fatal. Nausea and vomiting will be almost universal for persons in this group. The prodromal phase may also include prompt explosive bloody diarrhea, significant hypotension, and signs of necrologic injury. These patients should be sorted according to the availability of resources. Patients should receive symptomatic care. Lymphocyte analysis is necessary to support this classification.

e. These symptoms frequently occur in whole-body irradiated casualties within the first few hours of postexposure.

(1) *Nausea and Vomiting*. Nausea and vomiting occur with increasing frequency as the radiation exceeds 100-200 cGy. Their onset may be as long as 6-12 hours postexposure, but usually subside within the first day. The occurrence of vomiting within the first 2 hours is usually associated with a severe radiation dose. Vomiting within the first hour, especially if accompanied by explosive diarrhea, is associated with doses that frequently prove fatal. Due to the transient nature of these symptoms, it is possible that the patient will have already passed through the initial phase of gastrointestinal distress before being seen by a physician. It will be necessary to inquire about these symptoms at the initial examination.

(2) *Hyperthermia*. Casualties who have received a potentially lethal radiation injury show a significant rise in body temperature within the first few hours postexposure. Although the number of cases is few, this appears to be a consistent finding. The occurrence of fever and chills within the first day postexposure is associated with a severe and life-threatening radiation dose. Hyperthermia may occur in patients who receive lower but still serious radiation doses (200 cGy or more). Present evidence indicates that hyperthermia is frequently overlooked. Individuals wearing a chemical ensemble will normally be hyperthermic; consequently, this will not be a useful sign.

(3) *Erythema*. A person who received a whole-body dose of more than 1000-2000 cGy will develop erythema within the first day postexposure. This is also true for those who received comparable doses to local body regions, when the

erythema is restricted to the affected area. With doses lower but still in the potentially fatal range (200 cGy or more), erythema is less frequently seen.

(4) *Hypotension.* A noticeable and sometimes clinically significant decline in systemic blood pressure has been recorded in victims who received a supralethal whole-body radiation dose. A severe hypotensive episode was recorded in one person who had received several thousand rads. In persons who received several hundred rads, a drop in systemic blood pressure of more than 10% has been noted. Severe hypotension after irradiation is associated with a poor prognosis.

(5) *Necrologic Dysfunction.* Experience indicates that almost all persons who demonstrate obvious signs of damage to the central nervous system within the first hour postexposure have received a superlethal dose. Symptoms include mental confusion, convulsions, and coma. Intractable hypotension will probably accompany these symptoms. Despite vascular support, these patients succumb within 48 hours.

f. Casualties who have received a potentially fatal dose of radiation will most likely experience a pattern of prodromal symptoms that is associated with the radiation exposure itself. Unfortunately, these symptoms are nonspecific and may be seen with other forms of illness or injury, which may complicate the process of diagnosis. Therefore, the triage officer must determine the symptoms that have occurred within the first day postexposure, evaluate the possibility that they are indeed related to radiation exposure, and then assign the patient to one of the three categories: "Radiation Injury Unlikely"; "Radiation Injury Probable"; "Radiation Injury Severe." In the last two categories, the study of changes in circulating lymphocytes may either support or rule out the original working diagnosis. All combined-injury patients should be treated initially as if no significant radiation injury is present. Triage and care of any life-threatening injuries should be rendered without regard for the probability of radiation injury. The physician should make a preliminary diagnosis of radiation injury only for those patients for whom radiation is the sole source of the problem. This is based on the appearance of nausea, vomiting, diarrhea, hyperthermia, hypotension, and necrologic dysfunction.

618. Decontamination of Patient.

a. Radiation injury per se does not imply that the patient is a health hazard to the medical staff. Studies indicate that the levels of intrinsic radiation present within the patient from activation (after exposure to neutron and high-energy photon sources) are not life-threatening.

b. Patients entering a medical treatment facility should be routinely decontaminated if monitoring for radiation is not available. Removal of the patient's usually reduce most of the contamination. Washing exposed body clothing will surfaces will further reduce this problem. Both of these procedures can be performed in the field or on the way to the treatment facility. Once the patient has entered the treatment facility, care should be based on the obvious injuries. Care for life-threatening injuries should not be delayed until the decontamination procedures are completed.

c. When radiation safety personnel are available, decontamination procedures will be established to assist in rendering care and to minimize the hazard from radioactive contaminants. A more extensive decontamination procedure is to scrub the areas of persistent contamination with a mild detergent or a diluted strong detergent. Caution should be taken to not disrupt the integrity of the skin while scrubbing because disruption can lead to incorporation of the radioisotopes into deeper layers of the skin. Contaminated wounds should be treated first, since they will rapidly incorporate the contaminant. Washing, gentle scrubbing, or even debridement may be necessary to reduce the level of contaminants.

d. Wearing surgical attire will reduce the possible contamination of health personnel. If additional precautions are warranted, rotation of the attending personnel will further reduce the possibility of significant contamination or exposure. The prevention of incorporation is of paramount importance. The inhalation or ingestion of radioactive particles is a much more difficult problem, and resources to deal with it will not be available in a field situation.

619. Initial Treatment for Patients With Whole-Body Radiation Injury.

a. The primary determinants of survival among most patients receiving intermediate (serious but not uniformly fatal if treated) radiation doses is management of microbial infections and stopping any bleeding. If high intermediate doses have been received, fluid and electrolyte loss may cause early deaths. If properly resuscitated, however, these patients may survive until the consequences of hematologic failure become apparent.

b. For those casualties who have received sublethal whole-body radiation doses, gastrointestinal distress will predominate in the first 2 days. Antiemetics (metoclopramide, dazopride) may be effective in reducing the symptoms, but present drugs available have significant side effects. Unless severe radiation injury has occurred, these symptoms will usually subside within the first day. For those patients who continue to experience gastrointestinal distress, parenteral fluids should be considered. If explosive diarrhea occurred within the first hour postexposure, fluids and electrolytes should be administered if available. For triage purposes, the presence

of explosive diarrhea (especially bloody) is likely to be related to a fatal radiation dose.

c. Cardiovascular support for patients with clinically significant hypotension and necrologic dysfunction should be undertaken only when resources and staff allow. These patients are not likely to survive injury to the vascular and gastrointestinal systems combined with marrow aplasia.

620. Diagnosis and Treatment of Patient With Combined Injuries.

a. Conventional injuries should be treated first, since no immediate life-threatening hazard exists for radiation casualties who can ultimately survive. The patient with multiple injuries should be resuscitated and stabilized. During this process standard preparation for surgery will accomplish much radioactive decontamination. After surgery more definitive evaluation of radiation exposure can be initiated.

b. In the event of a radiation accident or nuclear detonation, many patients will probably suffer burns and traumatic injuries in addition to radiation. The initial triage of combined injury patients is based on these conventional injuries. Further reclassification may be warranted on the basis of prodromal symptoms associated with radiation injury. The prognosis for all combined injuries is worse than for radiation injury alone. Animal studies indicate that when other injuries are accompanied by sublethal doses of radiation, infections are much more difficult to control, and wounds and fractures heal more slowly. Thus, potentially survivable burns and trauma will be fatal in a large percentage of persons who have also received significant injury from sublethal doses of radiation. Often with conventional injuries, staged reparative surgery is scheduled for 1-2 days after the initial surgery, and reconstructive surgery is still later. Because of the delays in wound healing and the subsequent granulocytopenia and thrombocytopenia with injuries from nuclear weapons, most of the life-saving and reconstructive surgery must be performed within 36 hours after the exposure. Then, if possible, no surgery should be performed for the next 1 1/2-2 months postexposure.

621. Management of Infection.

a. In spite of antibiotics, infections with opportunistic pathogens are still a major problem. The majority of these organisms today are gram-negative like *Escherichia coli*, *Pseudomonas aeruginosa*, and many others. These infections occur as a consequence of both profound immunosuppression and abnormal colonization of body surfaces and invasive medical devices. Susceptible body surfaces include the oropharyngeal-respiratory tree and the intestine. Wound sites and artificial invasive devices such as catheters are also important sources of infection. Infections may be

more prevalent and severe if patients are maintained for long periods in environments containing antibiotic resistant pathogens.

b. Wound debridement, dressings, and, when necessary, antibiotics are key elements in infection control. Antibiotics, preferably in appropriate combination in therapy, should be used promptly to treat any new fever. When signs or symptoms of infection do appear in the granulocytopenic patient, treatment should be started without waiting for culture and sensitivity studies. Initial coverage should include gram-negative organisms and *Staphylococcus aureus*. Prevalent organisms and antimicrobial susceptibility patterns in the particular medical facility should also be considered. The drugs most often used now for the initial treatment are the synthetic penicillins, such as ticarcillin, combined with an aminoglycoside like tobramycin. It is recommended either that the treatment continue until the granulocytes return to more than 500 or treat for just 2 weeks and stop even if the white cell count is still low, as long as all signs of infection have cleared.

c. Systemic antibiotic therapy for management of infection is as follows.

(1) *Types of Agents*.

(a) Aminoglycosides such as gentarnicin, netilimicin, tobramycin, and amikacin are the most effective.

(b) Ureidopenicillins and carboxypenicillins such as ticarcillin and peperacillin are less effective than the aminoglycosides, but are synergistic with them against gram-negative enterics.

(c) Monobactams are effective against gram-negative enterics, to a lesser degree then aminoglycosides, but have no renal toxicity as they do.

(d) Beta Iactam resistant penicillins such as methicillin or dicloxicillin are effective for therapy of *Staphylococcus aureus*. Vancomycin can be administered for therapy of methicillin resistant *S. aureus*.

(e) Irnipenem (combined with cilastalin) is the only single agent that is effective against aerobic gram-positive and gram-negative organisms as well as anaerobic bacteria. However, some strains of *Pseudomonas* may be resistant.

(2) *Combination Therapy*. Several combinations have been advocated for the therapy of mixed aerobic-anaerobic infection, or for the therapy of gram-negative infections in the compromised host.

(a) *Gram-negative infection:* Aminoglycoside plus ureidopenicillins or carboxypenicillins; aminoglycoside plus a cephalosporin (second or third generation; arninoglycoside plus a monolactam.

(b) *Gram-positive infection:* Combinations of beta lactim resistant penicillin and an aminoglycoside.

(c) *Mixed aerobic-anaerobic infections:* An arninoglycoside or quinoline plus either clindamycin, cefoxitin, or metronidazole.

622. Future Concerns for Management of Radiation Injuries.

a. Treatable radiation-associated injuries only include those with the hematologic and possibly, the gastrointestinal syndrome. Combined injuries would shift the treatable range of injuries to the lower radiation doses. Even in these ranges there is very little definitive information available now. Many approaches suitable for conventional injuries may be found of little utility in irradiated subjects.

b. First actions in dealing with radiation casualties are to treat any conventional injuries first. Maintain ventilation and perfusion and stop hemorrhages. Most decontamination will be accomplished through routine management of the patient. Triage for radiation injuries followed by steps to prevent infection, fluid and electrolyte imbalance and bleeding will be necessary after patient stabilization. Unfortunately, there are limitations in the ability to effect these treatments successfully, particularly on a large scale with limited resources.

c. Presently new means of radioprotection and repair of radiation damage are on the horizon. Furthermore, immunomodulators are now under study which may not only facilitate marrow regeneration, but also help reduce the profound immunosuppression responsible for infections associated with severe injury. These agents may be used in combination with radioprotectors and antibiotics to further enhance survival. Leukopenia is a significant problem in irradiated casualties, but hazards exist with the transfusion of leukocytes into patients. Stem cell regeneration into selected populations probably offers the best opportunity to correct this deficiency. Although platelet transfusions are certainly desirable for radiation victims, they are presently not practical for mass casualty scenarios. A similar situation exists for bone marrow transplantation, although enormous progress is being made in autologous transplants. Again, stimulation of repair of surviving stem cells is probably the best near term hope of solving this problem. Problems of effective wound management and fluid and electrolyte replacement remain to be overcome in the neutropenic patient. Pharmacologic means to regulate performance decrements such as emesis and early transient incapacitation still are not available for use by military personnel.

d. The foregoing should clearly show that much remains to be done to achieve effective treatment of radiation or combined injury victims. However, progress in this area is being made and the concerns outlined above will be resolved.

623. Effect of Radiation Injury on Response to Trauma.

a. At Hiroshima and Nagasaki, large numbers of patients with traumatic injury developed complications 2 to 3 weeks after exposure which were characteristic of the effects of bone-marrow depression. The open wounds of many patients stopped healing and became hemorrhagic. There was an associated loss of granulation tissue. Patients lost weight, and many died as a result of overwhelming sepsis. Those patients who recovered went onto normal wound healing after return of bone-marrow function. This would be the typical clinical picture in patients exposed to prompt radiation from small weapons, while at the same time sustaining thermal or blast injuries. The most common form of radiation sickness would be the hematopoietic syndrome, and the resultant hemorrhagic tendencies and decreased resistance to infection would complicate the healing of these patients' wounds. The overall result would be prolonged hospitalization and increased morbidity and mortality.

b. Unfortunately, it will not always be possible at the time of admission to predict which patients with thermal or blast injuries would develop radiation sickness. A history of the prodromal symptoms which typically follow radiation exposure, as described previously, would be helpful but could not be relied upon. The first reliable indication that complications of radiation sickness might occur would be a lymphocytopenia, neutropenia, and thrombocytopenia noted in the peripheral blood count. By that time, however, the patient should have had at least the initial surgery required for his or her primary injuries. Subsequently, during the time the patients would be in the clinical phase of bone-marrow depression, careful supportive therapy would be required and elective surgical procedures should be avoided. Only those procedures that are actually required to save life and limb would be indicated. If surgery is required during the clinical phase of radiation sickness, increased morbidity and mortality would be expected. This could be minimized by applying the basic techniques of meticulous surgical care such as are commonly used in noncombat surgery on patients with hemorrhagic disorders.

c. Patients will also be seen who will have sustained their traumatic injuries and their exposures to radiation at different times. The best example of this would be patients wounded by conventional weapons before or after being exposed to fallout radiation. The interaction of the bone-marrow depression with traumatic injury is highly dependent upon this factor of timing. When patients are in the middle of the clinical phase of bone-marrow depression and are injured, the effect of this combination will be very deleterious, and a high mortality rate will be seen. If, on the other hand, the

clinical phase of sickness comes late in the course of wound healing, a relatively small interaction will be seen.

d. The degree of interaction between radiation sickness and traumatic injury will also depend a great deal upon the time course of the traumatic injury. Patients with small wounds that can be closed primarily, or with closed fractures which can be immobilized early, will not be as sensitive to the effects of radiation over as long a period of time as those patients with severe open wounds or burns. burn patients, in particular, will be susceptible to infection for an extended period of time and will be particularly sensitive to the decreased resistance to infection characteristic of radiation sickness. It will be expected then that the morbidity and mortality of burns combined with radiation sickness would be much greater than the morbidity and mortality following minor closed wounds and fractures. Open wounds and extensive soft tissue injuries would behave similarly.

e. Radiation injury can be combined with a number of other clinical problems. Radiation sickness may be superimposed on underlying medical diseases, and such patients will also be more sensitive to the deleterious effect of radiation sickness. There have been indications that radiation sickness will allow otherwise nonpathogenic bacteria to become pathogenic and to cause significant disease. Further, patients with mild radiation sickness which might otherwise go unrecognized would be much more sensitive to environmental stresses or to the effects of chemical and biological agents.

624. Effect of Injuries on Response to Radiation Sickness.

a. Many factors are responsible for relative radiation sensitivity. In any given population, some individuals will naturally be sensitive to irradiation and others will be relatively resistant. The factors which determine this are genetic as well as nongenetic. Age and physical condition are very important. The general condition of the individual at the time of exposure can modify the response to radiation considerably. There may be increased resistance to radiation if an individual has been exposed to a stressful stimulus such as a minor traumatic injury or environmental stresses prior to a radiation exposure. This phenomenon has been demonstrated in a number of laboratory studies with a number of animal species and a wide variety of stresses. Whether this applies to humans and to what degree is not known, but what can be said is that in combat situations that dose of radiation which would result in a given clinical response with a given probability is almost impossible to estimate.

b. An example of this problem is the question of the LD_{50} for people. A specific LD_{50} for individuals in combat cannot be given except as a broad range. The LD_{50} for a young adult unstressed and subject to a single acute exposure of gamma radiation

would probably be in the range of 450-500 cGy. There are indications that neutrons are more effective in producing lethality. (See paragraph 504b.) If the individual is stressed prior to radiation with a minor injury, the dose required to give a 50% probability of lethality may be increased by 50% or more. If on the other hand the radiation exposure is followed by some other injury, the dose which would result in a 50% mortality might very well be decreased by a factor of two. If an individual is exposed to a number of low dose rate, small exposures such as would occur from repeated entry into fallout fields, the dose required to result in 50% mortality would be increased.

c. If the factors of age, different physical conditions, etc., are added, and a large group of individuals are exposed to a variety of radiation exposures, combined or not combined with a variety of stresses and injuries, the result is a range for the LD_{50} that could be from as low as 200 to 450 cGy. This is an estimate, and proof of this will only come from actual combat experiences. If the exposure is a low dose rate exposure received over a long period of time (as in the case of fallout), the LD50 dose range could be considerably higher than 450 cGy. But variations such as this are quite possible and indicate why personnel dosimetry cannot be used as an absolute indication of prognosis. This is summarized in Figure 6-V.

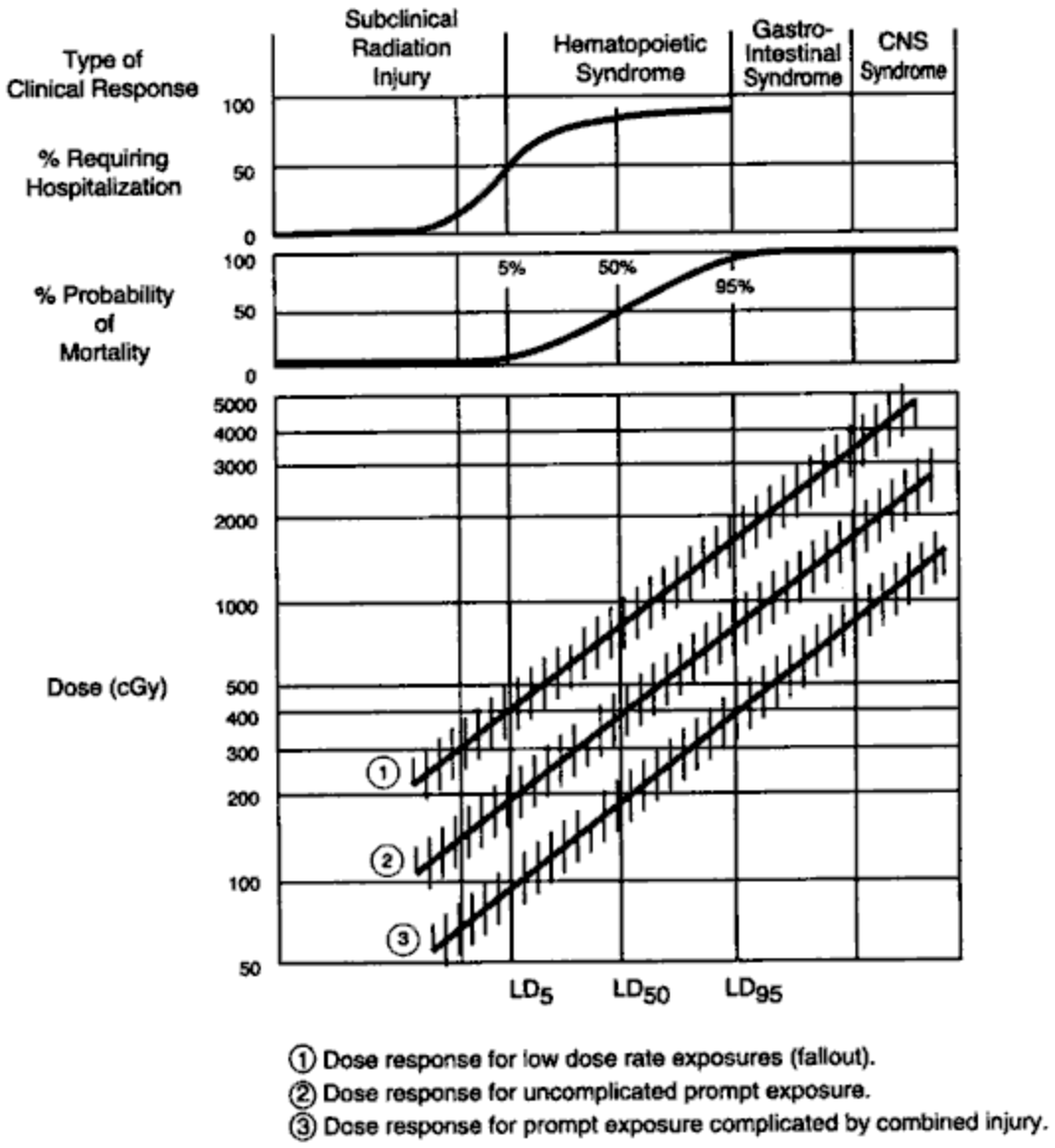


Figure 6-V. Clinical Effects of Whole Body Irradiation in Humans

d. Dosimetry for an individual patient should only be considered as an aid to diagnosis and prognosis. The patient's clinical condition combined with appropriate laboratory investigation will indicate the prognosis much better. It is perfectly possible for patients with a total exposure of 1000 rads or more, as recorded by personal dosimeters, to survive if that exposure was accumulated over a long period of time and particularly if it is not whole body and the patient is a young healthy adult.

SECTION V - PUBLIC HEALTH ASPECTS OF NUCLEAR WARFARE

625. Epidemic Disease Hazards Caused by Nuclear War.

a. For centuries the conduct and outcome of military operations have been profoundly affected by a small number of infectious diseases. The disruptive effects of war result in conditions conducive to increases in the incidence of these diseases, often in epidemic proportions. The use of nuclear weapons, with their potential for massive destruction, would produce situations in which epidemic outbreaks of disease among civilian populations would become highly probable. Enteric and respiratory diseases would be particular problems. These, in turn, could present serious hazards to military forces in the area and serious problems for a military medical service, particularly when civilian medical facilities and personnel are inadequate to handle the problems.

b. If large, heavily populated areas are devastated, the social organization which is required to effectively support a modem medical care system will be severely compromised. It is not until society reorganizes itself and rebuilds that a complex system such as modem medical care will resume. In past wars, military medical forces have provided for civilian populations and also the means for the rebuilding of a civilization ravaged by war. If the ravages of war are beyond the capabilities of either the society itself or the armies operating in the areas to repair, then the balance will be tipped in favor of decimation of the population by the classical diseases of disaster such as dysentery, typhus, typhoid fever, cholera, and plague. The results could be devastating to modem civilization.

626. Biomedical Impact of Nuclear Winter.

The concept of what has been termed "Nuclear Winter" is a rather recent concern. This is a phenomenon that has attracted much attention but little serious research. In the early 1980's the issue was politicized for various reasons. Therefore, a considerable amount of conjecture and hyperbole has surrounded the discussion of nuclear winter. However, there are certain phenomena that will be experienced in large amounts of dust, smoke, and debris injected into the upper atmosphere. This cloud would have a tendency to absorb or scatter the sunlight thus decreasing the surface temperature over a portion of the earth. This could conceivably interfere significantly with the production of foodstuffs in these regions. There is an additional concern that in the event of a high air burst, the nitrogen in the upper atmosphere would be converted into oxides and the oxides, in turn, would combine with the ozone layer thus depleting the protective ozone. This would cause a significant increase in the amount of ultraviolet light capable of reaching the earth's surface. The ozone layer would eventually be reestablished. The combination of cooling, decreased ambient light, and increased ultraviolet light bombardment could have a significant impact on food production and perhaps energy consumption. Serious research is needed to attempt to quantify these effects.

SECTION VI - PSYCHOLOGICAL ASPECTS OF NUCLEAR WARFARE

627. General.

Although it is possible to estimate roughly the number of injured and dead which would result from the thermal, blast, and radiation effects of a nuclear explosion, it is much more difficult to predict the numbers and types of psychiatric patients. It is generally felt that the types of acute psychological problems which would occur in such circumstances would be essentially the same as those seen in other combat situations, and that the treatment methods which have been developed as a result of experience in past wars would be appropriate.

628. Diagnosis.

a. The primary psychological abnormality which develops in severe stress or disaster situations is a transient, fluid state of emotional disruption. This occurs when individuals cannot cope with the danger presented to them by their environment. Its major features are fear and the results therefrom. The fear develops largely from the individual's inability to make meaningful decisions or initiate purposeful actions; and, as a result, even minor decisions become difficult to make. A vicious circle of fear-inaction-fear may ensue, and the individual involved may become ineffective. This may vary in degree all the way from very mild impairment of effectiveness to complete helplessness. Panic, defined as frantic, irrational behavior associated with real or supposed trapping, probably would be rare, since it has been found to be rare in other disaster situations. Precipitous flight with direction and purpose is not panic and should be considered a psychologically useful and practical response to the situation.

b. The characteristic disturbances which would occur include: stunned mute behavior, uncontrolled flight, tearful helplessness, apathetic depression, inappropriate activity, increased tension, or preoccupation with somatic representations. These disturbances can last for minutes, hours, days, or sometimes weeks. Longer term reactions may include phobias, survivor guilt, and psychosomatic symptoms. Fortunately, patients with the milder and shorter disturbances are in the majority.

629. Factors Determining Response.

The frequency and severity of the psychological disturbances vary with several factors.

a. *Intensity and Severity of Stress.* Stressful situations of brief duration are rather easily tolerated, and recovery of individuals with mild degrees of mental disruption under these circumstances is rapid. If stressful situations follow one another rapidly,

or if any one of them is of long duration, then the probability of the occurrence of more severe psychological reactions of longer duration increase.

b. *Degree of Personal Involvement.* If individuals have "close calls" or if they see close friends or relatives severely injured, their reactions will be more severe than if they remain relatively remote from danger.

c. *Degree of Training.* This is the most important factor in that it is one which is most easy to modify. Well-trained individuals, who can react readily to dangerous situations and initiate appropriate actions, will develop a minimum of incapacitating fear. The fear they do develop will, if anything, help them, since it will bean integral part of a reaction of increased awareness or alertness allowing more efficient fight or flight.

d. *Degree of Warning.* This is closely related to the above. Warning helps trained persons to prepare. They can initiate proper actions early. For untrained persons, the effect will be variable. If the fear is not incapacitating, then untrained persons who cannot react automatically to initiate proper actions may be able to utilize the time to improvise appropriate action. Whatever time they have to do this will help.

e. *Presence of Leadership.* In a disaster situation, a few individuals will emerge as leaders in a group. These may not be the appointed leaders, although in a military unit this is usually not the case unless the appointed or regular leaders become ineffective or are lost. When effective leadership is available, the group will fare much better than when there is none.

f. *Group Identification.* This is a particularly important factor for the military. If group or unit integrity is preserved, the individuals in the unit will do much better. Also, those individuals with mild psychological disruptions will recover faster if they can remain with or close to their unit, thus retaining their personal relationship as a member of the unit.

630. Treatment.

a. A major characteristic of these patients will be their suggestibility, and it is this which forms one of the basic underlying principles of treatment. The psychological disorders described do not require elaborate treatment and the best treatment is that which is simple, direct, and immediate. It should be done as far forward as possible, preferably within the unit to which the individual belongs. If this is not possible, then it should be started as soon as possible and in a medical facility close to the individual's unit. Evacuation to distant medical facilities is contraindicated. Evacuation tends to make the psychological problems worse by severing the patient's

relationship with his or her group or unit and by introducing the element of "secondary gain" with the removal of the patient from danger.

b. Treatment consists of:

(1) Reassurance and suggestion that the situation will improve. These people are suggestible early in their disruptive phase and simple reassurance using a positive, direct approach is usually successful. The individual should be made to feel that he or she has an excellent chance of recovery, which, in general, is true.

(2) Rest with removal from immediate danger. A short period of rest in a safe area is of great benefit.

(3) Catharsis. Retention of fear and anxiety by the more severely incapacitated frequently blocks effective communication: When the patient expresses his or her feelings, this tends to remove the block. This communication is essential before the individual can recover enough to rejoin the activities of his or her group or unit.

c. Psychiatrists are not always available to participate in the overall treatment of such patients. Therefore, all medical officers and their staffs should be familiar with these principles for managing the psychological problems arising from such disasters. The success of their actions will depend largely on how well the line commanders understand the program of managing this problem, since in a great degree the practical therapy of the mildly affected will be, in fact, the positive leadership actions taken by commanders.

631. Prevention.

The most important preventive factor is intensive training. The end result is less fear and more prompt effective action. Action relieves tension so that the fear response is less likely to become severe or incapacitating. Fear may not even develop to the point where the individual is aware of it. Other factors which contribute to prevention include discipline, morale, good leadership, and promotion of group identification. The beneficial results of effective command cannot be overemphasized.